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Air Pollution Assessment of Tetrachloroethylene

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Air Pollution Assessment of Tetrachloroethylene

B.B. FULLER

FEBRUARY 1976

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16. ABSTRACT

Tetrachloroethylene, commonly known as perchloroethylene, is a synthetic halogenated hydrocarbon with excellent solvent properties. The primary physiological effect of tetrachloroethylene inhalation is central nervous system depression with loss of consciousness and death occurring at high concentrations. Liver damage has also been reported but only at or near lethal levels.

The probability of environmental contamination is greatest at dry cleaning facilities where heated solvent vapors may be released to the atmosphere. Although adequate technology for the prevention of these losses is available, only 25% of the dry-cleaning establishments use such control. At metal degreasing facilities proper equipment design and adequate ventilation are sufficiently effective in maintaining safe vapor levels.

The high volatility and low solubility of tetrachloroethylene are responsible for the entry of the solvent into the atmosphere and the primary mode of transport for tetrachloroethylene photodegrades in sunlight with a half life of 2 days and is therefore not expected to accumulate in the environment. Concentrations in the ambient air over the highly industrialized Los Angeles Basin averaged 1.25 ppb but levels over rural areas and the open ocean averaged only 20 ppt.

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ABSTRACT

Tetrachloroethylene, commonly known as perchloroethylene, is a synthetic halogenated hydrocarbon with excellent solvent properties. Although about 60% of domestic consumption of tetrachloroethylene is by the dry cleaning industry, tetrachloroethylene is also used for both vapor and cold degreasing of metals. Workers at dry-cleaning and metal degreasing facilities are subject to tetrachloroehtylene exposure and numerous episodes of both acute and chronic exposure have been reported. The primary physiological effect of tetrachloroethylene inhalation is central nervous system depression with loss of consciousness and death occurring at high concentrations. Liver damage has also been reported but only at or near lethal levels.

The probability of environmental contamination is greatest at dry cleaning facilities where heated solvent vapors may be released to the atmosphere. Although adequate technology for the prevention of these losses is available, only 25% of the dry-cleaning establishments use such control. At metal degreasing facilities proper equipment design and adequate ventilation are sufficiently effective in maintaining safe vapor levels.

The high volatility and low solubility of tetrachloroethylene are responsible for the entry of the solvent into the atmosphere and the primary mode of transport for tetrachloroethylene is expected to be atmospheric. However, tetrachloroethylene photodegrades in sunlight with a half life of 2 days and is therefore not expected to accumulate in the

environment. Concentrations in the ambient air over the highly industrialized Los Angeles Basin averaged 1.25 ppb but levels over rural areas and the open ocean averaged only 20 ppt. Even at the highest concentrations observed, the amount of tetrachloroethylene present was too small to constitute any hazard to health.

ACKNOWLEDGEMENTS

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TABLE OF CONTENTS

	Page
LIST OF FIGURES	viii
LIST OF TABLES	ix
I. SUMMARY AND CONCLUSIONS	1
II. AIR POLLUTION ASSESSMENT REPORT	7
A. PHYSICAL AND CHEMICAL PROPERTIES	7
B. EFFECTS	13
1. Human	13
2. Animal	28
3. Other Effects	47
C. AMBIENT CONCENTRATIONS, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY	49
D. SOURCES	69
E. CONTROL	78
REFERENCES	81

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LIST OF FIGURES

Figure Number		Page
I	TETRACHLOROETHYLENE EXPIRED AIR CONCENTRATIONS FOLLOWING VAPOR EXPOSURE	19
II	EVAPORATION RATE OF $Cl_2C=C\,Cl_2$ FROM WATER	50
III	MAP OF THE LOS ANGELES BASIN	55
IV	CONCENTRATION VS. TIME DATA FOR C_2Cl_4 AT GROUND LEVEL AT LOCATION 43 IN THE WEST SAN GABRIEL VALLEY	60
V	TYPICAL CONCENTRATIONS OF HALOCARBONS AND AMBIENT TEMPERATURE VS. ALTITUDE OVER INGLEWOOD	61
VI	LOCATIONS OF UNITED STATES TETRACHLOROETHYLENE PRODUCTION FACILITIES	71
VII	PRODUCTION AND SALES OF TETRACHLOROETHYLENE (1940-1972)	73
VIII	SUMMARY OF PRODUCT RELEASE INFORMATION	77

LIST OF TABLES

Table Number		Page
I	PHYSICAL PROPERTIES OF TETRACHLOROETHYLENE	8
II	CONCENTRATION OF TETRACHLOROETHYLENE IN FOOD STUFFS	14
III	OCCURRENCE OF TETRACHLOROETHYLENE IN HUMAN TISSUE	16
IV	EFFECTS OF SINGLE EXPOSURES OF TETRACHLOROETHYLENE AT VARYING CONCENTRATIONS IN HUMANS	21
V	TOXICOLOGICAL EFFECTS	31
VI	DECOMPOSITION RATES OF ETHYLENE IN THE DARK AND IN THE PRESENCE OF SUNLIGHT	53
VII	CONCENTRATIONS OF TETRACHLOROETHYLENE AT VARIOUS LOCATIONS IN LOS ANGELES COUNTY	56
VIII	OCCURRENCE OF TETRACHLOROETHYLENE IN THE ENVIRONMENT	64
IX	U.S. TETRACHLOROETHYLENE PRODUCTION CAPACITY (1975)	72
X	CONSUMPTION OF TETRACHLOROETHYLENE	76

AIR POLLUTION ASSESSMENT OF TETRACHLOROETHYLENE

I. SUMMARY AND CONCLUSIONS

Tetrachloroethylene, commonly called perchloroethylene, is a clear, colorless, volatile, nonflammable liquid. It is an excellent solvent for a variety of organic substances. Tetrachloroethylene is the most chemically stable of all chlorinated ethylenes. It decomposes in contact with active carbon, at high temperatures (700°C), or high pressure (30,000 atm, 350°C), and photodegrades in sunlight with a half-life of two days. Products of photolysis include Cl_2, HCl, and trichloroacetic acid. Tetrachloroethylene undergoes autooxidation in the presence of oxygen and ultraviolet light to give trichloroacetyl chloride and the highly toxic compound, phosgene, but this reaction can be prevented by small (<1 percent by weight) additions of stabilizers. Due to its high vapor pressure (20 mmHg at 26°C) and low water solubility (0.015 g/100 g), the primary mode of transport for tetrachloroethylene is expected to be atmospheric.

Tetrachloroethylene is fatal to animals when ingested in large doses (5 ml/kg for mice and rabbits), but the major causes for concern are the nonlethal adverse effects due to inhalation. The primary physiological effect as a result of acute or chronic inhalation is central nervous system depression. Symptoms include lightheadedness, dizziness, sleepiness, impaired motor coordination, nausea, lassitude, tremors, headache, loss of appetite, and dissorientation. These symptoms have been observed at concentrations as low as 77 ppm.

Loss of consciousness and death, possibly due to depression of respiration or cardiac failure, occur at high concentrations (1500 to 2000 ppm). Evidence of liver damage from both chronic and acute exposures has also accumulated. Fatty degeneration, necrosis, and cirrhosis of the liver, as well as altered liver function tests have been observed in man as well as other species. Guinea pigs exhibit particular sensitivity to tetrachloroethylene. Symptoms of liver dysfunction appear after exposure to concentrations as low as 100 ppm, while chronic exposure to about 300 ppm allegedly produced similar symptoms in an occupationally exposed human. Tetrachloroethylene is stored in the lipid portion of fatty tissue and released and metabolized at a slow rate. Therefore, a brief overexposure may subject organs such as the liver to continuous chemical insult over a period of weeks, and an acute exposure may have a chronic effect.

Other toxic effects, including renal damage, depressed antibody formation, inhibition of growth, loss of sense of smell, and gastric hemorrhage, have been observed in animal studies and select medical cases. Evidence for central nervous system effects is well documented, reproducible and, in general, conclusive. Evidence for liver toxicity is conflicting. At similar concentrations with similar durations of exposure, some studies have reported effects, while others have claimed no effect in the identical species. Exposure levels resulting in liver damage are generally close to levels resulting in death. Therefore, liver damage is only a problem at or near lethal levels and cannot be realistically considered a major toxicological threat to humans. No evidence of carcinogenicity, mutagenicity, or teratogenicity for tetrachloroethylene has been reported in humans. Exposure of pregnant mice and rats to 300 ppm on days 6 to 15 of gestation did not result in teratogenesis. No differences in the incidence of tumors were observed between control and experimental rats exposed to 300 or 600 ppm of tetrachloroethylene.

The high volatility and low solubility of tetrachloroethylene are responsible for the entry of the solvent into the atmosphere. Although release to the hydrosphere may occur, rapid evaporation (50 percent in 26 minutes) is expected. Oxidative decomposition in water occurs with a half-life of 8.8 months. Thus, minimal long-term accumulation in the hydrosphere is realized.

Approximately 6.5×10^8 lbs (3.25×10^5 tons) of tetrachloroethylene were consumed domestically in 1974 and 85 percent of this amount (2.76×10^5 tons) was lost to the atmosphere. Concentrations of the solvent detected at points far removed from the source of contamination provide evidence for the contention that aerial transport is the major dispersal route.

Levels of tetrachloroethylene in the ambient air over the highly industrialized Los Angeles Basin have been reported. Concentrations there averaged 1.25 ppb, with a maximum of 4.2 ppb (1974). A progressive decrease in concentration was noted upon moving from the inland valley to the coast and was interrupted only by high concentrations in the Los Angeles business district. Concentrations appear dependent upon the time of day and are consistent with diurnal wind patterns of that area. In this basin, there is a possibility that, during summer months, when stagnant inversion conditions are present on consecutive days, a greater buildup of tetrachloroethylene may occur. Ambient tetrachloroethylene levels over rural areas and the open ocean average about 20 ppt.

The highest concentrations to which an average person might be exposed is expected to occur in the vicinity of production plants or industrial workshops where the solvent is used. Although ambient concentrations of tetrachloroethylene near these facilities have not been reported, estimates based on production figures and hypothetical plant conditions indicate that ambient concentrations 500 meters downwind of a production facility would be 406 μg/m, or 0.06 ppm. Workers at dry cleaning and metal degreasing facilities are particularly subject to tetrachloroethylene exposure and numerous episodes of both acute and chronic exposure have been reported.

The most sensitive and precise method for analysis of tetrachloroethylene is gas chromatography-mass spectrometry (sensitive to 5 ppt). Solvent vapors may be trapped efficiently with activated charcoal. This collection method allows frequent sampling of air. Hence, areas where solvent levels may be high (e.g., industrial workshops) can easily be subjected to comprehensive sampling and measurement programs. The present allowable time-weighted average levels over a typical workday (8 hours) are set at 100 ppm by the American National Standards Institute.

Tetrachloroethylene is a synthetic halogenated hydrocarbon. Approximately 735 million pounds (3.68×10^5 tons) were produced in the United States in 1974, out of a world production capacity estimated at close to 1×10^6 tons. Currently, the major process for synthesizing tetrachloroethylene consists of the thermal chlorination of propane. About 60 percent of domestic consumption of tetrachloroethylene is by the textile industry (primarily for dry cleaning). This solvent commands 80 percent of the dry cleaning market and is used by 55 percent of the dry cleaning plants in the United States. Tetrachloroethylene utilization is second to trichloroethylene for the vapor degreasing of metals. It is also used for cold degreasing. As a chemical intermediate, tetrachloroethylene is used in the synthesis of Fluorocarbons 113-116.*

Production of tetrachloroethylene by thermal chlorination of propane is conducted in a closed system. The probability of environmental contamination resulting in the production stages is viewed as minimal. The major loss of tetrachloroethylene to the environment occurs in dry cleaning operations where heated solvent

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*Fluorocarbon 113--trichlorotrifluoroethane
Fluorocarbon 114--dichlorotetrafluoroethane
Fluorocarbon 115--chloropentafluoroethane
Fluorocarbon 116--hexafluoroethane

vapors may be released to the atmosphere. This loss, however, may be prevented by use of a water-cooled condenser system which recovers 95 percent of the vaporized solvent and by adsorption of the remainder on activated carbon. Approximately 25 percent of the dry cleaning establishments use such control. Vapor degreasing operations also experience tetrachloroethylene loss to the environment. However, vapor reclaiming devices, such as cooling coils, can minimize the loss. Adequate ventilation; proper use of equipment; and prompt cleanup of leaks, spills, and waste receptacles all contribute to the maintenance of safe ambient levels in the workshop environment.

In view of the available evidence, tetrachloroethylene does not appear to be a serious environmental residual. Under laboratory conditions and accidental direct inhalation, its chief physiological impact in both chronic and acute dosages is central nervous system depression. There is some evidence suggesting liver damage as well. Even at the highest concentrations observed in the environment, the amount of tetrachloroethylene present was too small to constitute any hazard to health. Since the compound is fairly rapidly degraded in the environment and no evidence of significant bioaccumulation exists, tetrachloroethylene does not represent a significant pollution threat to the general population.

II. AIR POLLUTION ASSESSMENT REPORT

A. PHYSICAL AND CHEMICAL PROPERTIES

Tetrachloroethylene is a clear, colorless, volatile, dense liquid with a pleasant ethereal odor. It is nonflammable and noncombustible. It is the most chemically stable (thermally and photolytically) of all chlorinated ethanes and ethylenes. However, in the absence of small quantities of stabilizers, tetrachloroethylene is corrosive to metals. It is an excellent solvent for a large number of organic substances, including fats, oils, tars, rubber, and gums.

Tetrachloroethylene possesses both a high vapor pressure and low water solubility (see Table I). Therefore, its primary mode of environmental transport is expected to be the atmosphere. Since it has a vapor density 5.5 times greater than that of air, it might tend to spread laterally instead of upward under stagnant conditions (2). Physical properties of tetrachloroethylene are presented in Table I.

Tetrachloroethylene dissolves sulfur, iodine, mercuric chloride, and aluminum chloride. At room temperature, it dissolves 0.4 percent (by weight) of ammonia, and is a solvent for benzoic, cinnamic, phenylacetic, phenylpropionic and salicylic acids. However, it does not dissolve sugar, glycerol, or protein to an appreciable extent.

Stabilized tetrachloroethylene can be used in the presence of air, water, and light and with common construction metals at temperatures less than 140°C. In the absence of catalysts, moisture, and oxygen, the compound is stable to about 500°C. At 700°C, it decomposes in

TABLE I

PHYSICAL PROPERTIES OF TETRACHLOROETHYLENE

Structural formula:		$Cl_2C = CCl_2$
Molecular weight:		165.83
Freezing point:		-22.7°C
Normal boiling point:		121.2°C
Dielectric constant		2.353 at 15°C 2.134 at 90°C
Dipole moment:		0
Refractive index:		1.5055
Latent heat of vaporization at normal boiling point:		50.0 cal/gm
Latent heat of fusion:		2.525 kcal/g-mole
Thermal conductivity,	liquid:	0.0732 BTU/(hr)(ft^2)(°F/ft)
	vapor:	0.00505 BTU/(hr)(ft^2)(F/ft) at 121.2°C
Specific heat, calories,		
	liquid:	0.205 at 20°C/(gm)(°C)
	vapor:	0.146 at 100°C, 1/atm
Solubility in water:		0.015 gm/100 gm at 25°C
Solubility of water in solvent:		0.008 gm/100 gm
Solubility in octanol:		infinite
Partition coefficient water/air:		1.22 at 20°C
Log partition coefficient octanol/water:		2.86
Fire point:		nonflammable
Flash point:		nonflammable
Vapor density:		5.8 gm/l 760 mm Hg at 121.2°C

TABLE I (Continued)

Viscosity, vapor, 60°C: 0.0099 centipoise*

Vapor pressure:

°C	mmHg	°C	mmHg
-20.6	1	70	155.3
+ 2.4	5	80	226.0
13.8	10	90	319.2
26.3	20	100	438.5
40.0	41	110	591.6
50.0	67	121.2	760.0
60.0	104		

Density, liquid:

°C	d^t_4** (g/cc)
10	1.63120
15	1.63109
20	1.62260
30	1.60640
120	1.44865

Viscosity, liquid:

°C	centipoise**
15	0.932
20	0.880
25	0.839
30	0.798
50	0.657
75	0.534

---

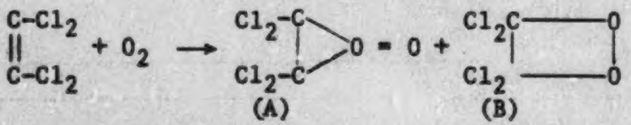
[*] 1 centipoise = 0.01 g/cm sec

[**] Density at the given temperature related to that of water at 4°C (1.000).

contact with active carbon to hexachloroethane and hexachlorobenzene. The same reaction takes place at 350°C under very high pressure (30,000 atm.)

The unstabilized compound, when in contact with moisture, slowly decomposes to yield trichloroacetic and hydrochloric acids.

Tetrachloroethylene photodegrades in sunlight with a half-life of two days (76). Products of photolysis include free chlorine (32.5 percent of theoretical), hydrogen chloride (16.4 percent), and trichloroacetic acid (36.4 percent) (2). Tetrachloroethylene is unaffected by oxygen in the absence of light. When irradiated with ultraviolet light in the presence of oxygen, tetrachloroethylene undergoes auto-oxidation to trichloroacetyl chloride. An intermediate stage in this reaction is the formation of peroxy compounds.



Compound (A) undergoes rearrangement to give trichloroacetyl chloride and oxygen. Compound (B) decomposes to two molecules of phosgene.* This slow decomposition can be prevented by the addition of stabilizers such as acetone, acetylenic compounds, aniline, borate esters, n-butane, o-cresol, diisopropylamine, ethyl acetate, hydrazine derivatives, isobutyl alcohol, lactones, o-nitrophenol, pyrazoles, stearates or SO_2. Tetrachloroethylene reacts rapidly with hydroxyl ions (half-life of 8 days) and slowly with alkyl peroxy radicals and ozone (half-lives of 220 and 11 years, respectively) (3). With ozone, it decomposes to a mixture of phosgene and trichloroacetyl chloride. Oxidizing tetrachloroethylene with a mixture of sulfuric and nitric acids yields predominantly trichloroacetyl chloride, with some tetrachlorodinitroethane as a by-product. Similar yields are also obtained with fuming nitric acid. Tetrachloroethylene reacts readily with nitrogen dioxide at 100°C to yield tetrachlorodinitroethane. It may also be partially oxidized with sulfur trioxide at 150°C to yield trichloroacetylchloride.

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*While a discussion of phosgene, otherwise known as carbonyl chloride ($COCl_2$), is beyond the scope of this report, it should be mentioned that the compound is a suffocating and highly poisonous war gas and is much more hazardous than its precursor.

Total decomposition of tetrachloroethylene to HCl and elemental carbon occurs at 220°C in the presence of excess hydrogen and a reduced nickel catalyst. The expected reaction under these conditions is reduction at the double bond. At high pressure, tetrachloroethylene is completely decomposed by ammonia to ammonium chloride and elementary carbon.

Tetrachloroethylene reacts explosively with butyl lithium in petroleum ether. Explosion also occurs with molten potassium, but not with sodium. In the presence of dibenzoyl peroxide, tetrachloroethylene is reported to give copolymers with styrene, vinyl acetate, methyl acrylate and acrylonitrile. Research progress has shown that tetrachloroethylene undergoes photochemical degradation under simulated tropospheric conditions. A significant study is currently being conducted by D. Lillian at Rutgers State University in New Jersey (4). The project, entitled "Photochemical and Atmospheric Reactions," is examining the chemical and physical nature of photochemical smog. Simulated sunlight is being used to statically and dynamically irradiate synthetic atmospheres. The kinetics and mechanisms of utilization and formation of major reactants, products and intermediates are being measured.

B. EFFECTS

1. Human

Vapor phase tetrachloroethylene poses the greatest toxic threat to humans because it is most readily absorbed through the lungs and only slightly from the gastrointestinal tract (5). The fact that tetrachloroethylene has been used safely and effectively in rather large doses (4 doses of 4 cc each) as an anthelmintic* in humans testifies to the relative safety of ingestion (5,6,7). The concentrations of tetrachloroethylene in a variety of foodstuffs are presented in Table II and give some indication of the quantities of this solvent being ingested by the general population.

Several studies on the fate of inhaled tetrachloroethylene have been reported. Ogate _et_ _al_. (8) exposed 13 volunteers of average age 34.5 years to 87 ppm for 3 hours and subsequently measured excretion of their water-soluble metabolites. Excretion of trichloroacetic acid (TCA) increased progressively from the start of exposure until 3 hours following exposure and decreased to normal in about 64 hours. Only 1.8 percent of the intake amount was excreted as TCA in 67 hours (2.8 days), with an additional 1.0 percent excreted as an unidentified chloride. It was concluded that tetrachloroethylene was partially eliminated through exhalation, but mostly retained in the body during the first 67 hours. These results were verified by later studies:

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*An agent that is destructive to worms.

TABLE II

CONCENTRATION OF TETRACHLOROETHYLENE IN FOODSTUFFS

FOODSTUFF	TETRACHLOROETHYLENE µg/kg
Dairy Produce	
Fresh milk	0.3
Cheshire cheese	2
English butter	13
Eggs	ND
Meat	
English beef (steak)	0.9
English beef (fat)	1.0
Pig's liver	5
Oils and Fats	
Margarine	7
Olive oil (Spanish)	7
Cod liver oil	2
Vegetable cooking oil	0.01
Castor oil	3
Beverages	
Canned fruit drink	2
Light ale	ND
Canned orange juice	ND
Instant coffee	3
Tea (packet)	3
Wine (Yugoslav)	ND
Fruits and Vegetables	
Potatoes (S. Wales)	ND
Potatoes (N.W. England)	0.7
Apples	2
Pears	2
Tomatoes*	1.2
Black grapes (imported)	ND
Fresh bread	1

ND - not detected

* Tomato plants were grown on a reclaimed lagoon at Runcorn Works of ICI.

Source: McConnell, G., D.M. Ferguson and C.R. Pearson, "Chlorinated Hydrocarbons and the Environment," Endeavour 34, 13-18, 1975.

Bolanowska and Golacka (9) exposed male subjects to 390 mg/liter tetrachloroethylene for 6 hours with two 30-minute breaks. Elimination via exhaled air amounted to 25 percent of intake; percutaneous* excretion was insignificant (0.02 percent of the intake/hour), urinary excretion was less than 10 percent, and 62 percent was retained in the body. Tada and Nakaaki (10) exposed 2 male subjects repeatedly for 2 to 4 hours per day to 50 to 160 ppm tetrachloroethylene and studied the relationship of urinary TCA levels to ambient concentrations of vapor and exposure duration. Net excretion of TCA increased daily, reaching a maximum in three to four days. Daily excretion rates were reasonably linear, both with ambient tetrachloroethylene levels and exposure times.

Excretion of metabolites in workers occupationally exposed to tetrachloroethylene was studied by Ikeda and Imamura (11) and Ikeda et al. (12). Urine samples were collected from 85 workers in 17 workshops where the vapor concentrations were fairly constant. Samples were analyzed for TCA and total trichloro-compounds. Urinary metabolite levels were seen to increase until the atmospheric concentration of the solvent reached 50 to 100 ppm. Further metabolite increases were not observed beyond this concentration. The mean biological half-life of tetrachloroethylene was found to be 144 hours (3 to 4 times longer than trichloroethylene). This suggests that tetrachloroethylene is retained in the body for long periods. Table III presents the results of postmortem analyses of human tissue for tetrachloroethylene.

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*Performed through the skin.

TABLE III

OCCURRENCE OF TETRACHLOROETHYLENE IN HUMAN TISSUE

Age of Subject	Sex	Tissue	Concentration (in µg/kg wet tissue)
76	F	Body fat	6
		Kidney	<0.5
		Liver	<0.5
		Brain	<0.5
76	F	Body fat	1
		Kidney	6
		Liver	2
		Brain	<5
82	F	Body fat	0.4
		Liver	1.2
48	M	Body fat	0.8
		Liver	0.7
65	M	Body fat	21
		Liver	3.4
75	M	Body fat	29.2
		Liver	4.3
66	M	Body fat	0.5
74	F	Body fat	4

Source: McConnell, G., D.M. Ferguson, and C.R. Pearson, "Chlorinated Hydrocarbons and the Environment," Endeavour 34, 13-18, 1975.

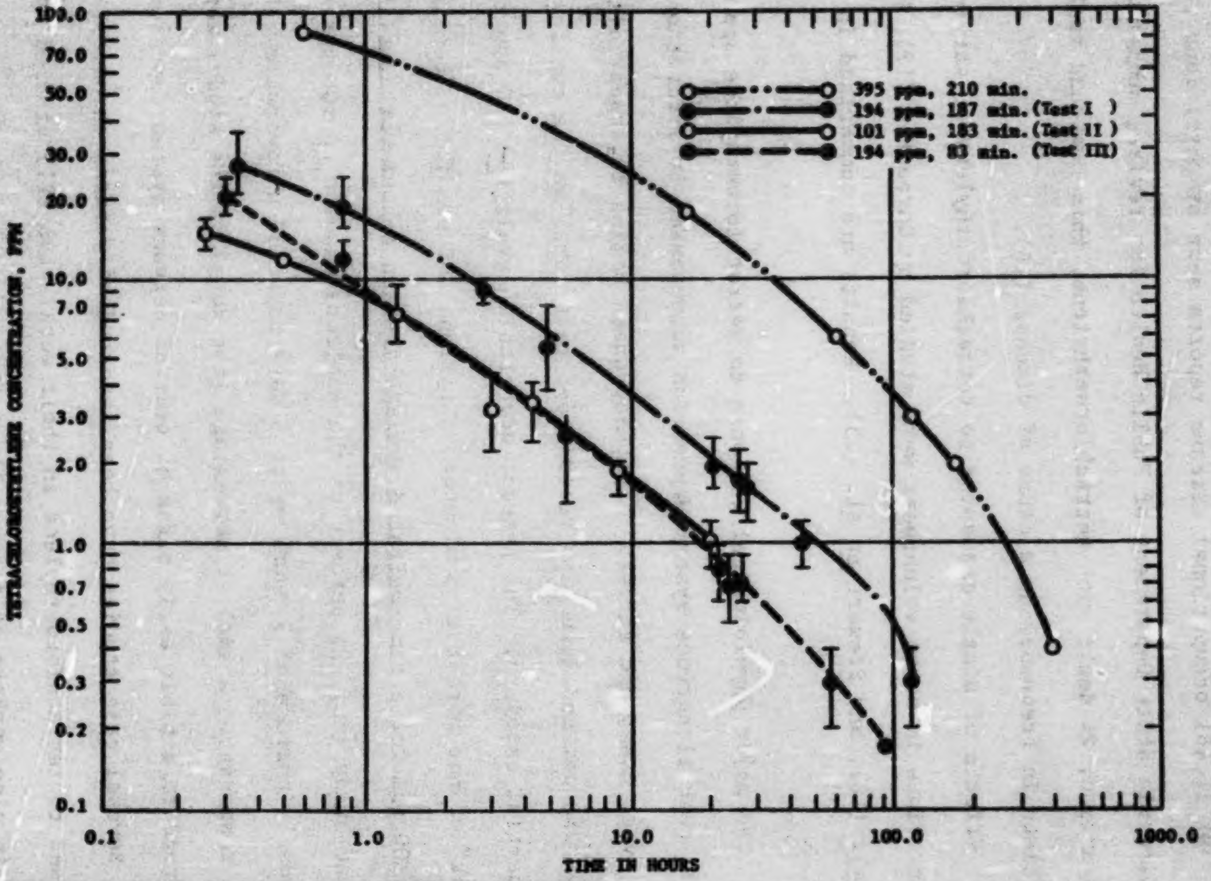
Tetrachloroethylene metabolites can be detected in the urine, even though they are present in small quantities. From certain studies (11,12) it appears that metabolite concentrations are proportional to exposure concentrations only at low ambient levels. Consequently, in the case of a suspected overdose, it would be difficult to determine the extent of exposure by urinalysis. In contrast, most volatile solvents (including tetrachloroethylene) absorbed by the body circulate through the bloodstream and are excreted, in part, by the lungs. Both qualitative and quantitative estimates of the extent of exposure can be determined by analysis of a sample of expired air. Infrared spectrographic methods (sensitivity, 2 ppm) provide a characteristic spectrum of the compound. Vapor phase chromotography (VPC) provides a very sensitive method (0.01 ppm), which can be used for post-exposure analysis of residual solvent levels. Stewart _et al_. (13) demonstrated the usefulness of breath analysis as a means of determining exposure levels. Male volunteers, aged 30 to 59, were exposed as indicated below:

Test	Number of Subjects	Time (minutes)	Concentration Range (ppm)	Total Exposure (ppm)
I	6	187	106-224	194
II	6	83	93-225	194
III	6	183	59-147	101

Blood, urine, and expired air concentrations were determined and correlated with the magnitude of the exposure. No tetrachloroethylene was detected in the urine during and after exposure in either Test

I or II. Blood concentrations during exposure to 194 ppm increased from 0 to about 2.6 ppm after 3 hours (Test I), but the compound was no longer detectable in the blood 30 minutes following exposure. As indicated in Figure 1, tetrachloroethylene possesses an exceedingly prolonged decay in the post-exposure expired air (14). The compound was detectable in the expired air 94 hours after an 83-minute exposure to 194 ppm (Test II). The average concentration in the expired air was proportional to the level of exposure when the durations of exposure were identical (Tests I and III). Decay curves of similar slope resulted from exposures of similar duration. The length of time that the compound was measurable in the expired air was proportional to both the vapor concentration and the length of exposure. No overlap in expired air concentration occurred with ambient levels of 100 ppm and 200 ppm for a duration of 3 hours (Tests I and III). Thus, a subject's breath analyzed 100 hours after an alleged 3-hour exposure to tetrachloroethylene could be used to determine ambient levels during this exposure. The opportunity to test this hypothesis arose when a case of gross overexposure (average: 395 ppm, 3.5 hours) was examined (serial samples of expired air were analyzed over a 400-hour period). Results are presented in Figure 1. Tetrachloroethylene was detectable in the expired air 400 hours (~17 days) following exposure. It is believed that the long excretion or decay curve of tetrachloroethylene in the breath is due to the fact that this compound is stored in the lipid and released and metabolized at a very slow rate (15).

FIGURE 1
TETRACHLOROETHYLENE EXPIRED AIR CONCENTRATIONS FOLLOWING VAPOR EXPOSURE



Source: Stewart, R. D., D. S. Arley, A. W. Schaffet and H. H. Gay. "Accidental Exposure to Anesthetic Concentrations of a Solvent Containing Tetrachloroethylene." Ind. Med. and Surg. 30 327-330 (1961).

Of 16,461 occupational disease reports sent by physicians to the California State Department of Public Health for review, summarization, and action, 24 dealt with tetrachloroethylene, this compound ranking fiftieth in frequency as a cause of disease (16).

Effects of single exposures to tetrachloroethylene at varying concentrations in human volunteers were studied by Carpenter (17), Rowe _et al_. (18), and Stewart _et al_. (13). Results are summarized in Table IV.

The major physiological response to tetrachloroethylene appears to be central nervous system depression accompanied by irritation of the eyes, nose, and throat. Lightheadedness begins to appear slightly above 200 ppm and Rowe (18) recommended this level as the ceiling for industrial exposure. The present acceptable levels are 300 ppm for a "peak" of not more than 5 minutes' duration, 200 ppm for a "ceiling," and 100 ppm for a time-weighted average over an 8-hour workday (19). In one study (20), 25 percent of the subjects exposed to 100 ppm for daily periods of 7 hours up to 5 days reported unfavorable subjective responses. A smaller percentage (not noted in the study) were observed to exhibit early signs of central nervous system depression.

Several case studies have been reported of persons chronically exposed to tetrachloroethylene in their work. Lob (21) studied symptoms in nine workers at a tetrachloroethylene production facility. Two workers showed severe neurological disturbances including tremors,

TABLE IV

EFFECTS OF SINGLE EXPOSURES OF TETRACHLOROETHYLENE AT VARYING CONCENTRATIONS IN HUMANS

CONCENTRATION (ppm)	LENGTH OF EXPOSURE	EFFECTS	TIME TO RECOVER
50	immediate	Odor apparent.	—
75-80	1-4 minutes	Slight, passing eye irritation consisting of mild burning sensation.	
100-120	1 hour	Feeling of "fullness" in the head. Slight soft palate irritation accompanied by dryness; mild eye irritation attributed to transient concentration peaks. No effect on motor coordination.	—
200-240	0.5-2 hours	Stinging in eyes persisted after 30 minutes. Congestion of frontal sinuses with slight nasal discharge; slight lightheadedness, dizziness and sleepiness.	1 hour
280	2 hours	Lightheadedness, burning in eyes, congestion of frontal sinuses, thickness of tongue, tightness about mouth, irresponsibility, nausea, impaired motor coordination.	1 hour
475-600	2 hours	Increased salivation, sweetish metallic taste, irritation to the eyes, tightness in frontal sinuses, increased perspiration of hands, increased secretion of mucous from the nasal passages, slight nausea, dizziness, numbness about mouth, loss of inhibition. Mental effort needed for good motor coordination.	1 hour

TABLE IV (Continued)

CONCENTRATION (ppm)	LENGTH OF EXPOSURE	EFFECTS	TIME TO RECOVER
1000	1.5 hours	Marked irritation to eyes and upper respiratory tract noted after 1 min.; dizziness noted after 2 mins.; tightness of frontal sinuses, lassitude, mental fogginess, anesthetization of lips and tip of nose and definite exhilaration noted by 45 mins. Increased perspiration of hands, irritation around nostrils, congestion of the eustachian tubes.	—
raised from 1000 to 1500	immediate	Loss of inhibition, faintness and dizziness, aching of facial muscles, nausea. Dyspnea upon exertion, mental sluggishness, slight inebriation and a slight effect upon physical balance noted following exposure.	—
2000	immediate	Anesthesia	—

— No recovery time reported.

loss of equilibrium, difficulty in walking, and abnormal sensory reactions. Seven workers, employed from three weeks to eight months, complained of less severe symptoms such as weakness, dizziness, headache, nausea, vomiting, anorexia,* and irritability. Air concentrations at the plant were only 75 ppm.

Gold (22) reported the case of a 47-year-old man who had operated a dry cleaning establishment using tetrachloroethylene for 3 years. His chronic symptoms were similar to those reported by Lob and persisted for 12 months following termination of exposure. When admitted to the hospital, he was seen to have acute conjunctivitis of the right eye and loss of sense of smell on the right side. His ability to concentrate was markedly impaired. His symptoms were suggestive of both cerebral-cortical damage** and basal ganglia involvement***.

Evidence for liver damage as a result of chronic tetrachloroethylene exposure has also accumulated. Coler and Rossmiller (23) reported seven cases of industrial exposure during tank-cleaning operations. Concentrations ranged from 230 to 385 ppm and occurred 2 days per week over a period of from 2-1/2 to 6 years. All seven workers exhibited the usual symptoms of malaise, dizziness, headache, irritation of nasal mucosa, light-headedness, nausea, fatigue, staggering gait, stomachache, slowed ability to think, memory impairment, and a drunkenlike state. In addition, one man had a severe gastric hemorrhage and cirrhosis of the liver.* Three others exhibited signs of liver dysfunction, including altered sulfobromophthalein** retention time and altered serum protein patterns.

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*Loss of appetite.

**Damage to the convoluted layer of gray substance that covers each hemisphere of the cerebrum.

***Masses of gray matter centrally embedded in the cerebral hemisphere.

Trense and Zimmerman (24) reported the case of a 23-year-old man who had worked for over a year, once again, in a dry cleaning establishment where tetrachloroethylene was used. This individual died two days following hospital admission from cardiovascular failure. Autopsy and chemotoxic findings showed toxic liver cell necrosis and toxic myocardial fatty degeneration. Quantities of chlorinated hydrocarbon metabolites were detected in the cerebrum but not in the lungs, liver, kidney, or blood. Death was ascertained to be due to chronic tetrachloroethylene exposure in the absence of adequate ventilation.

Cases of acute exposure in which the tetrachloroethylene came into direct contact with the skin are numerous (25-28). The results of such exposure are erethema*** and blistering.

Absorption through the skin is not a serious problem. Stewart and Dodd (29) submerged the thumbs of 5 subjects for 40 minutes in tetrachloroethylene and measured the post-exposure breath concentration.

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*A disease of the liver marked by progressive destruction of liver cells accompanied by degeneration of the liver substance and increase of connective tissue.

**An increase in retention time above the normal range is indicative of liver dysfunction.

***Redness of the skin produced by congestion of the capillaries.

The mean peak concentration was 0.31 ppm; the mean concentration 2 hours following exposure was 0.23 ppm. Of all the solvents tested, tetrachloroethylene had both the slowest rate of skin penetration and the slowest decay in the expired breath. It was concluded that tetrachloroethylene is not likely to be absorbed in toxic quantities when in contact with the skin of the hands and forearms.

The primary physiological response to acute vapor exposure in the absence of skin exposure is also central nervous system depression, leading to loss of consciousness (14, 30, 31, 33) and, in several instances, death (21, 32). Damage to kidney and liver have been known to accompany acute as well as chronic exposures (30). While the compound does not possess a great potential for causing serious liver or kidney injury, a subclinical hepatitis* may occur. The SGOT** and SGPT** concentrations are useful indicators of the induced hepatocellular injury as is the urinary urobilinogen level, which is seen to rise seven to ten days following exposure. However, the absence of abnormal liver function indices in exposed individuals should be viewed with caution. Increased blood serum levels of enzymes, such as the transaminases, are an indication of liver cell necrosis, which usually accompanies pronounced fatty degeneration of the liver. This accumulation of liver fat is believed to be due to inhibited synthesis of the protein component of the lipoprotein, the form in which triglycerides are released from the liver. It is possible that the hepatotoxic effect of tetrachloroethylene is lipoprotein synthesis inhibition, while leaving the cellular structure intact. Since fairly substantial fatty degeneration may occur without pathological values showing in conventional hepatic function tests, damage could result in animals and man that would only be observed upon autopsy. Since fatty degeneration of long duration is thought to be capable of leading to cirrhosis of the liver, concentrations of tetrachloroethylene which are capable of producing fatty degeneration should be prohibited (34). Acute renal failure and hypotension* probably occur secondary to central nervous system depression, the former as a consequence of the shock produced by peripheral vascular collapse and the latter following myocardial anoxia** due to depression of respiration.

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*Slight inflammation of the liver.

**Serum glutamic-oxalacetic transaminase; serum-glutamic-pyruvic transaminase. Increased levels are indicative of liver damage.

Liver damage was reported in two similar instances where exposure resulted from the use of tetrachloroethylene for cleaning purposes in a poorly ventilated area. In one case (30), the transient, minimal liver damage was denoted by slight elevations in SGOT levels on the second and third post-exposure days and an increased urinary urobilinogen on the ninth day. In the second case (14), elevated total serum bilirubin and alkaline phosphatase were observed as well, on the ninth and fourteenth post-exposure days, respectively. The urinary

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*Lowered blood pressure.
**Reduction of oxygen in the heart below physiological levels.

urobilinogen returned to the normal range by the fourteenth post-exposure day. Of 9 firemen called to investigate a tetrachloroethylene leak at a dry cleaning establishment and exposed to concentrated fumes for only 3 minutes, 8 showed liver damage as seen by elevated SGOT levels 12 days after exposure. One of these also showed an enlarged spleen and liver. All returned to normal within 22 to 63 days. It should be noted at this point that an acute exposure to tetrachloroethylene may, in fact, represent a chronic exposure due to retention of the compound in the body. Since the compound is stored in the lipid and released and metabolized at a slow rate, a brief overexposure may subject organs, such as the liver, to continuous chemical insult over a period of two weeks. Thus "an acute exposure to this solvent (tetrachloroethylene) has a chronic effect upon the liver and is capable of producing a mild hepatitis" (14).

Pulmonary edema* was reported in two instances of exposure to tetrachloroethylene. In one case (33), the patient recovered fully after several weeks; in the second, the patient died (21).

Not all cases of unconsciousness, liver damage, and death are the result of occupational exposure to select populations. With the advent of coin-operated dry cleaning machines, tetrachloroethylene has found its way into the public domain. In 1965, Braddock reported the death of a 16-year-old boy from tetrachloroethylene intoxication due to a contaminated sleeping bag (32). No evidence of carcinogenicity, mutagenicity, or teratogenicity for tetrachloroethylene has been reported.

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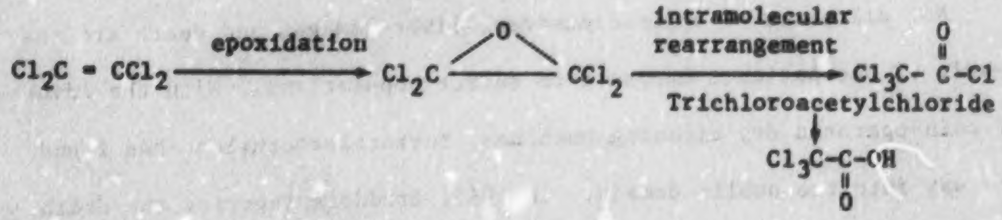
*An effusion of fluid into the air vesicles and interstitial tissue of the lungs.

2. Animal

In animals, just as in humans, tetrachloroethylene is absorbed mainly through the lungs. Cats and young dogs absorb more through the gastrointestinal tract than mature dogs, but mice and rabbits absorb it most easily of all by this route (5). Orally administered, tetrachloroethylene is used as an anthelmintic in dogs (35, 36), being 90 percent effective at a dose of 3 mg/dog against Ancylostoma canium.

Rats and mice were shown to excrete trichloroacetic acid and trichloroethanol following inhalation of 200 ppm of tetrachloroethylene for 2 hours (37). Yllner exposed mice to ^{14}C-tetrachloroethylene and noted that 52 percent of the total urinary radioactivity was in the form of trichloroacetic acid (38). He suggested the formation of an epoxide, which was further converted to trichloroacetyl chloride, as a possible metabolic route.



It is important to note here the potential mutagenicity and carcinogenicity of epoxides with specific reference to tetrachloroethylene oxide postulated in the previously noted scheme (39).

Also, as in man, tetrachloroethylene is stored in the rat lipids. Jacobs *et al.* (40) fed rats a mixture of 7 chlorinated hydrocarbon solvents, including tetrachloroethylene, for 4 to 12 weeks. All seven solvents were detected in the abdominal and renal adipose tissue.

The extent to which bioconcentration of tetrochloroethylene occurs in rainbow trout muscle was studied by Neely *et al.* (77). The rate constant for uptake was determined by measuring the concentration in fish muscle at various time intervals following the placement of the fish in a water bath containing trace amounts of tetrachloroethylene. The rate constant for clearance was determined by a similar procedure after the fish were placed in freshwater. The ratio of these two constants provides an estimate of the bioconcentration factor at a steady rate.

Rate constant for uptake (k_1) = 3.323 ± 0.45 hours^{-1}
Rate constant for clearance (k_2) = 0.0823 ± 0.030 hours^{-1}
Bioconcentration $\left(\frac{k_1}{k_2}\right) = 39.6 \pm 5.5$
Bioconcentration $\left|\log\left(\frac{k_1}{k_2}\right)\right| = 1.59\ (1.4\text{-}1.74)$

By testing a variety of chemicals in this manner, it was shown that the bioconcentration potential of a chemical can be predicted with reasonable accuracy if the octanol/water partition coefficient is known. The logarithm (log) of the partition coefficient is directly proportional to the bioconcentration factor over a wide range of values (77).

Tetrachloroethylene, if given in sufficiently high doses, can be lethal to a variety of species regardless of the route of administration. Levels resulting in death to 50 percent of the test animals are summarized in Table V under each species for which such data have been reported.

The effects of wide ranges of concentrations on the central nervous system in rats and other species have been reported under both chronic and acute conditions. In a recent study by Leong *et al.* (41), two groups of rats, consisting of 192 animals each (96 male and 96 female), were exposed respectively to 300 and 600 ppm of tetrachloroethylene vapor for 6 hours per day, 5 days per week, for 12 months. No signs of central nervous system depression were observed during these exposures. These results confirmed an earlier study by Rowe *et al.* (18) in which a maximum of 179 seven-hour exposures to 400 ppm produced no observable effects in rats, rabbits, and monkeys, respectively. Eighteen, seven-hour exposures to 1600 ppm produced an interesting syndrome in rats (18). During the first week, they were seen to become drowsy and stuporous; during the second week, they exhibited marked salivation, extreme restlessness or nervousness, stupor, disturbance of equilibrium and coordination, a biting reflex upon contact with another rat, and a scratch reflex. These symptoms were apparently due to irritation of the cholinergic nervous tissue.* At 250 ppm, rats

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*Those nerve fibers which liberate acetyl choline at a synapse when a nerve impulse passes.

TABLE V

TOXICOLOGICAL EFFECTS

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Mouse	Intra-peritoneal	3.2ml/kg; 2.9ml/kg	1 dose	50% death in 24 hours	46, 73
Mouse	Oral	5.0ml/kg	1 dose	50% death in 24 hours	74
Mouse	Inhalation	35.4mg/liter	24 hours	50% death	75
Mouse	Inhalation	3700 ppm	730 min	50% death in 24 hours	
Mouse	Inhalation	3700 ppm	24 min	anesthetization	45
Mouse	Inhalation	3700 ppm	470 min	liver dysfunction	
Mouse	Intra-peritoneal	24-28 mmoles/kg	1 dose	liver dysfunction; enlargement of hepatocytes; vacuolization; slight necrosis	46, 45
Mouse	Inhalation	200 ppm	4 hrs/day, 6 days/week, 1-8 weeks	fatty degeneration of the liver	34
Mouse	Intra-peritoneal	2.5-2.9 ml/kg	1 dose	kidney dysfunction	46
Mouse	Inhalation	300 ppm	7 hours/day, days 6-15 of gestation	increased liver weight; decreased fetal body weight; increased subcutaneous edema (fetal toxicity); minor skeletal abnormalities	2

TABLE V (Continued)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Mouse	Inhalation	200-1600 ppm	8 hrs/day, 3 days	increase in mortality and inhibition of growth	51
Mouse	Inhalation	0.1-0.5 mg/liter	5 hrs/day, 3 months	decreased electroconductance of muscle and amplitude of muscular contraction	53
Mouse	Subcutaneous injection	1.5ml/kg	alternate days for 20-40 days	decreased serum albumin level, increased γ globulin levels	54
Rat	Inhalation	1600 ppm	18 7-hr exposures	drowsiness, stupor, increased salivation, extreme restlessness, disturbance of equilibrium and coordination, biting and scratching reflex	18
Rat	Inhalation	2500 ppm	1-13 7-hr exposures	loss of consciousness & death	18
Rat	Inhalation	600 ppm	6 hrs/day, 5 days/week, 12 months	increased mortality over controls	41
Rat	Inhalation	230 ppm	8 hrs/day, 5 days/week, 7 months	congestion and light granular swelling of kidneys	17
Rat	Inhalation	470 ppm	8 hrs/day, 5 days/week, 7 months	increased secretion, cloudy swelling and desquamation of kidneys, congestion and cloudy swelling of liver; congestion of spleen	17

TABLE V (Continued)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Rat	Inhalation	3000-6000 ppm	up to 8 hours	increase in liver weight, increase in total lipid content of liver accompanied by a few diffusely distributed fat globules	18
Rat	Inhalation	300mg/m^3	entire gestation period	decreased levels of DNA and total nucleic acids in the liver, brain, ovaries and placenta	50
Rat	Inhalation	300 ppm	7 hr/day, days 6-15 of gestation	decreased maternal weight gain; increased incidence of fetal resorption	2
Rabbits	Oral	5cc/kg	1 dose	death within 24 hours	5
Rabbits	Inhalation	2500 ppm	28 7-hr exposures	central nervous system depression without unconsciousness	18
Rabbits	Inhalation	15 mg/1	45 days	liver damage indicated by elevated SGPT, SGOT, SGLDH; marked reduction of Schmidt index	42
Rabbits	Inhalation	100 mg/m^3	3-4 hrs daily, 7-11 months	moderately increased urinary urobilinogen; pathomorphological changes in the parenchyma of liver and kidneys	43

TABLE V (Continued)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Rabbits	Inhalation	15 mg/1	45 days	significant reduction of glomerular filtration rate and the renal plasma flow; decrease of highest excretory tubular capacity (kidney damage)	44
Rabbit	Inhalation	100mg/m^3	3-4 hrs/day, 7-11 months	depressed agglutinin formation	43
Rabbit	Inhalation	15mg/1	45 days	increased plasma levels of adrenal cortical and adrenal medullar hormones; increased excretion of principal catecholamine metabolite	52
Guinea Pig	Inhalation	2500 ppm	18 7-hr exposures	loss of equilibrium, coordination and strength	18
Guinea Pig	Inhalation	400 ppm	169 7-hr exposures	increase liver weight, increase in neutral fat and esterified cholesterol in the liver and moderate central fatty degeneration with slight cirrhosis	18
Guinea Pig	Inhalation	100 ppm	132 7-hr exposures	slight increase in liver weight, in lipid content and several small fat vacuoles in liver	18

TABLE V (Continued)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Flat Fish	Dissolved in water	5 mg/1	96 hours	lethal to 50% of fish	59
Cat	Oral	4cc/kg	36 hours	death	5
Dog	Intraperitoneal injection	2.1ml/kg	24 hours	lethal to 50% of animals	48
Dog	Intraperitoneal injection	7.4mmole/kg	24 hours	liver dysfunction (as measured by increased SGPT levels in 50% of animals)	48
Dog	Intraperitoneal injection	14mmole/kg	24 hours	kidney dysfunction (as measured by increased retention of PSP in 50% of animals)	48
Human	Oral	3 ml	single dose	mild transient gastrointestinal reactions	6
Human	Inhalation	75-80 ppm	1-4 min.	slight transient eye irritation	13, 17, 18
Human	Inhalation	100-120 ppm	1 hour	soft palate irritation accompanied by dryness; mild eye irritation	13, 17, 18
Human	Inhalation	1500 ppm	immediate	faintness; aching facial muscles; dyspnea upon exertion, mental sluggishness, slight inebriation and slight effect upon physical balance noted after exposure	13, 17, 18

TABLE V (Continued)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Human	Inhalation	475-600 ppm	2 hours	increased salivation, increased perspiration of hands, increased secretion of mucous from the nasal passages, numbness about mouth, mental effort needed for good motor coordination	13, 17, 18
Human	Inhalation	1000 ppm	1 hr.,35 mins.	marked irritation to eyes and upper respiratory tract; lassitude, mental fogginess, anesthetization of lips and tip of nose, exhilaration, congestion of the eustachian tubes	13, 17, 18
Human	Inhalation	230-385 ppm	2 days/week, 2 1/2-6 years	severe gastric hemorrhage and cirrhosis of the liver; signs of liver dysfunction	23
Human	Inhalation	high	1 year	hemoptysis, coughing, sweat attacks, jaundice, oliguria, hematemesis, cardiovascular failure and death	24
Human	Inhalation	high	actue	unconsciousness; death	14, 21, 27, 28, 30, 31, 32, 33

TABLE V (Concluded)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Human	Skin contact	liquid	30 mins.	burns, erythema, blistering	27, 28
Human	Inhalation	high	acute	transient liver damage	14, 30, 31
Human	Inhalation	high	acute	pulmonary edema	21, 33

experienced severe central nervous system depression with loss of consciousness and death in from 1 to 13 seven-hour exposures. Rabbits were less affected, exhibiting central nervous system depression with no unconsciousness after 28 seven-hour exposures. Loss of equilibrium, coordination, and strength were observed in guinea pigs after 18 seven-hour exposures.

The primary acute toxic effects observed in rats were severe central nervous system depression characterized by varying degrees of "drunkenness," stupor, and unconsciousness, or death resulting from failure of respiration or possibly cardiac function (18). After 14 hours at 2000 ppm, neither death nor unconsciousness was observed. Effects on the central nervous system were apparent after 0.2 hours at 3000 ppm, but several hours were necessary to induce unconsciousness. No deaths were observed at 4 hours, but 20 percent of the animals died within 8 hours. Unconsciousness resulted after several minutes at 6000 ppm; no deaths were observed at 0.6 hours, but 8 hours at this concentration proved fatal to 85 percent of the animals.

In the study by Leong _et al_. (41) described previously, the animals were observed for physiological changes both during and following their 12-month exposure. When they became moribund or died, autopsy was performed. No adverse effects of exposure were observed with respect to growth, demeanor, hematology, urinalysis, or gross pathology. However, starting from the fifth month of exposure, the mortality of male rats exposed to 600 ppm of tetrachloroethylene was significantly higher than that of control rats. Male rats exposed to 300 ppm or female rats exposed to either 300 or 600 ppm showed no difference in mortality. The cause of this increased mortality remains unknown.

Slight disagreement exists between this study and one performed much earlier by Carpenter (17). In the latter study, 24 rats were exposed to averages of 70, 230, and 470 ppm for 8 hours a day, 5 days per week, over a period of 7 months. At 70 ppm, no toxic effects were observed. However, at 230 ppm, some animals showed congestion of the kidneys and light granular swelling but no sign of progressive injury to the liver. At 470 ppm, the liver was congested, and cloudy swelling was apparent. Increased secretion, cloudy swelling, and desquamation* were observed in the kidneys, and congestion was noted in the spleen as well. An interesting observation was that the exposed animals showed a stimulation of fertility, with more litters being conceived, all of above-average size.

Liver and kidney damage in rabbits chronically exposed to tetrachloroethylene has also been observed. Rabbits exposed to concentrations of 15 mg/l for a period of 45 days experienced elevated serum GPT, GOT, and GLDH* activities after 30 days, indicating release of both cytoplasmic (GPT) and mitochrondical (GOT and GLDH) enzymes from the liver and, thus, liver damage. Marked reduction of the Schmidt Index

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*The shedding of epithelial elements, chiefly of the skin, in scales or sheets.

**Glutamic dehydrogenase.

(GPT and GOT/GLDH) indicated a prevalence of mitochondical over cytoplasmic lesions (42). Rowe (18) observed slight degeneration of the liver of rabbits following 28 seven-hour exposures to 2500 ppm tetrachloroethylene. Navrotskii et al. (43) reported moderately increased urinary urobilinogen and pathomorphological changes in the parenchyma* of both the liver and kidneys after 7 to 11 months at 100 mg/m^3 for 3 to 4 hours daily.

Renal clearances of endogenous creatine and paramino-hippuric acid were studied in 12 rabbits exposed to 15 mg/l tetrachloroethylene for 45 days. Statistically significant reduction of the glomerular** filtration rate and the renal plasma flow and a significant decrease of the highest excretory tubular capacity were observed. Renal tubular function was altered more than glomerular function (44).

Guinea pigs appear to be particularly sensitive to the hepatotoxic effects of tetrachloroethylene. Following 169 seven-hour exposures to 400 ppm tetrachloroethylene, guinea pigs exhibited fatty degeneration of the liver with slight cirrhosis. Rats, rabbits, and monkeys were not affected by similar treatment (18). A statistically significant increase in lipid content and several small fat vacuoles were observed in guinea pigs after 132 seven-hour exposures to concentrations as low as 100 ppm (18).

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*A term used to designate the functional elements of an organ as distinguished from its framework.

**The glomeruli of the kidney are two coils of blood vessels, one projecting into the expanded end of each of the uriniferous tubules (channels for the passage of urine).

Gehring (45) studied the hepatotoxic potency of tetrachloroethylene relative to its narcotic and lethal potencies in mice. At 3700 ppm, the LT_{50}* was 730 minutes; the ET_{50}** for anesthetic action was 24 minutes; and the ET_{50} for hepatic dysfunction, as measured by a significantly elevated SGPT, was 470 minutes. Thus, the duration of exposure needed to cause elevation of the SGPT level is considerably larger than that needed to produce anesthesia and was long enough to produce death in a number of animals.

Both Gehring (45) and Klaassen and Plaa (46) studied the relative doses needed to induce liver dysfunction (as measured by elevated SGPT levels) and death in mice via intraperitoneal injection. Gehring found the 24-hour LD_{50}*** to be 32.2 mmole/kg****. The 24-hour ED_{50}***** for elevation of the SGPT level was 24.0 mmole/kg. The ratio $LD_{50}/ED_{50} = 1.43$. Klassen and Plaa found the 24-hour LD_{50} to be 28 mmole/kg; the 24-hour ED_{50} for elevation of the SGPT level was also 28 mmole/kg. Here, near lethal doses were necessary before liver dysfunction became apparent. Pathological examination revealed enlargement of hepatocytes with cellular infiltration and vacuolization and slight necrosis* when concentrations were in the lethal range.

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*The length of time at a given exposure level in which 50 percent of the animals tested die.

**The effective time at which the desired result is observed in 50 percent of the animals tested.

***The dose lethal to 50 percent of the animals tested.

****mmole/kg: millimole per kilogram.

*****The effect dose at which the desired result is observed in 50 percent of the animals tested.

Kylin _et al_. (34) studied the hepatotoxicity in mice of inhaled tetrachloroethylene over long periods (chronic exposure). Four groups of 20 mice each were exposed for 4 hours daily, 6 days per week, to 200 ppm tetrachloroethylene for 1, 2, 4, or 8 weeks. Examination of the liver disclosed massive infiltration of fat, usually central and involving at least one-half of the lobules. The fatty degeneration tended to be more severe with longer exposure and resulted in a twofold increase in liver fat.

Kidney dysfunction induced by intraperitoneal injection in mice was studied by Plaa and Larson (47) and Klaassen and Plaa (46). In both studies, near lethal doses (2.5 to 2.9 ml/kg) were required before any impairment became apparent. At 2.5 ml/kg, 17 percent of the animals showed over 100 mg percent protein in the urine, one animal out of 6 showed less than 50 percent of the proximal convoluted tubules to be necrotic, 4 out of 6 showed more than 50 percent swelling, and 2 out of 6 showed less than 50 percent swelling.

The relative effects of tetrachloroethylene, administered intraperitoneally, on liver and kidney function in dogs was also studied by Klaassen and Plaa (48). The 24-hour LD_{50} was found to be 21 mmole/kg.

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*Death of tissue usually as individualized groups of cells or in small localized areas.

Chronic exposure of rats to higher concentrations of tetrachloroethylene was studied by Rowe et al. (18). Eighteen 7-hour exposures to 1600 ppm resulted in a slight depression of growth and enlargement of the liver and kidneys, but without accompanying histopathological changes. Cloudy swellings and a few small fat vacoules appeared in the livers of rats after thirteen 7-hour exposures to 2500 ppm.

Acute exposure of rats to high concentrations was also studied by Rowe (18). Rats that survived exposures causing high mortality (3000 and 6000 ppm for up to 8 hours) showed minor changes in the liver, including slight increase in weight, slight increase in total lipid content, and slight cloudy swellings accompanied by a few diffusely distributed fat globules. Rats exposed to concentrations which did not result in any deaths (2000 ppm for up to 14 hours) showed similar symptoms after 5 hours of exposure. Single exposures resulting in no organic injury were 0.2 hours at 12,000 ppm, 0.4 hours at 6000 ppm, 3 hours at 2500 ppm, and 5 hours at 1600 ppm. At 0.6 hours, 5.0 hours, and 7.0 hours, organic damage was observed at each of the above concentraions, respectively. Ikeda et al. (49) also observed no changes in rats exposed to 1080 ppm for 4 hours. Carpenter reported that those rats which survived 19,000 ppm for more than 30 minutes showed congestion and granular swelling of the liver, while those surviving 9000 ppm for more than 3 hours showed similar changes in the liver and also marked granular swelling of the kidneys (17).

The 24-hour ED_{50} for the appearance of liver dysfunction (as measured by increased SGPT levels) was 7.4 mmole/kg. The 24-hour ED_{50} for the appearance of kidney dysfunction (as measured by retention of PSP*) was 14 mmole/kg. The ratio LD_{50}/ED_{50} (PSP) = 1.5; the ratio LD_{50}/ED_{50} (SGPT) = 2.8. Thus, near lethal doses are required to produce kidney dysfunction in dogs; however, liver dysfunction appears at much lower doses in this animal.

By comparison with animal studies, it is possible to calculate the exposure necessary to produce fatty degeneration in the livers of exposed humans. However, the figure will differ depending upon which species is chosen for comparison. Guinea pigs, highly susceptible to hepatotoxic chemicals, show significant fatty degeneration after 169 seven-hour exposures at 400 ppm (18). This is equivalent to an industrial worker breathing four times the allowed level seven hours per day, five days per week, for about eight months. If lower levels over a more prolonged time cause the same effect, then this same worker will show evidence of fatty degeneration after 3 years at 150 ppm. However, if the rat is chosen for comparison, the most recent study (41) indicates that no pathological changes were observed in the livers of animals exposed to 600 ppm for one year.

Several studies on the effects of tetrachloroethylene exposure on pregnant animals have been reported. Exposure of female rats to 300 mg/m^3 (44 ppm) for the entire gestation period caused a decrease in the levels of DNA and total nucleic acids in the liver, brain, ovaries, and placenta (50). Exposure of pregnant mice and rats to 300 ppm for 7 hours a day on days 6 through 15 of gestation resulted in a decrease in maternal weight gain among rats, an increase in the relative weight of the liver in mice, an increase in the incidence of fetal resorption of rats, decreased fetal body weight of mice, and an increased incidence of subcutaneous edema (fetal toxicity) and minor skeletal anomalies in mice. No teratogenic effects were observed in either species at this level of exposure (2).

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*Phenolsulfonephthalein. Failure to excrete the normal amounts in a given time is indicative of liver damage.

Exposure of three-week-old mice for 8 hours a day, 3 days each, to 200, 400, 800, and 1600 ppm caused inhibition of growth and significant increase in mortality (51).

Various miscellaneous physiological effects have also been reported. When rabbits were exposed to 100 mg/m^3, 3 to 4 hours per day, for 7 to 11 months, agglutinin* formation was depressed after one to 1-1/2 months. No effects were observed at 10 mg/m^3 (43). Plasma levels of adrenal cortical and adrenal medullar hormones gradually increased during exposure of 10 rabbits for 45 days to concentrations of 15 mg/l. Urinary excretion of 3-methoxy-4-hydroxy mandelic acid, the principal catecholamine metobolite, also increased slightly during this period. The variations appear to be related to the stimulatory action of tetrachloroethylene on the adrenal capsules (52).

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*Antibody which aggregates a particulate antigen, such as bacteria, following combination with the antigen.

The electroconductance in the gastrocnemius muscle of the right rear extremity of mice exposed to narcotic doses of tetrachloroethylene for one to 2 hours decreased by 10 to 15 percent. Subnarcotic doses resulted in a three to four percent decrease. Prolonged inhalation (0.1 or 0.5 mg/1, 5 hours daily for 3 months) decreased the electroconductance by 5 to 10 percent and the amplitude of muscular contractions by 8, 12, and 24 percent after 1, 2, and 3 months, respectively. One month after termination of exposure, the electroconductance of the gastrocnemius muscle was at the control level (53). Subcutaneous injections of 1.5 ml/kg body weight every other day for 20 to 40 days markedly decreased serum albumin content and increased γ-globulin content in mice (54).

No instances of mutagenicity or of teratogenicity have been reported. The studies by Leong _et al_. (41) discussed previously were basically intended to evaluate the carcinogenic potential of tetrachloroethylene. In this study, no discernible difference in the incidence of observed tumors between the exposed and control rats was noted. The study, entering its 24th month, will continue until all the animals have died in order to maximize the opportunity for a cancer-producing effect to be manifested. The toxicological effects on experimental animals and humans are summarized in Table V.

3. Other Effects

Tetrachloroethylene was observed to stop growth in the bacteria *Streptococcus faecalis* (55) and to inhibit sporulation in *Monilinia fructicola* (56). It was also seen to be effective as a vermifuge. That is, it caused *Polydora websteri* and nereid worms to emerge from oyster shells (57).

Tetrochloroethylene is not expected to undergo bacterial biodegradation since no microbial BOD_5* was observed (76).

Ongoing research into the health effects of tetrachloroethylene include the following studies:

- The studies reported by Leong *et al*. (41) in Section II.B.2., concerning the carcinogenic potential of tetrachloroethylene over the life span of the rat following a one-year exposure to 300 and 600 ppm, are presently in their 24th month. As no increase in the incidence of tumors has yet been observed, the study will continue until all of the rats have died, at which time a final report including a statistical analysis of the data will be presented. The studies are being conducted at the Toxicology Research Laboratory, Health and Environmental Research, the Dow Chemical Company, Midland, Michigan.

- The National Cancer Institute is currently conducting a study of the potential carcinogenicity of tetrachloroethylene administered by stomach tube in rats (58).

- A project entitled, "Behavioral/Neurological Evaluation of Solvent Exposures," seeks to (1) detect behavioral and neurological changes associated with chronic occupational exposure to low levels of toxic solvents; (2) correlate such changes with indices of ambient exposure, body burden, and records of accidents and illnesses; and (3) apply and use behavioral function tests as early warning indicators of overexposure. More than 450 workers, each exposed to one of four solvents (including tetrachloroethylene), will be

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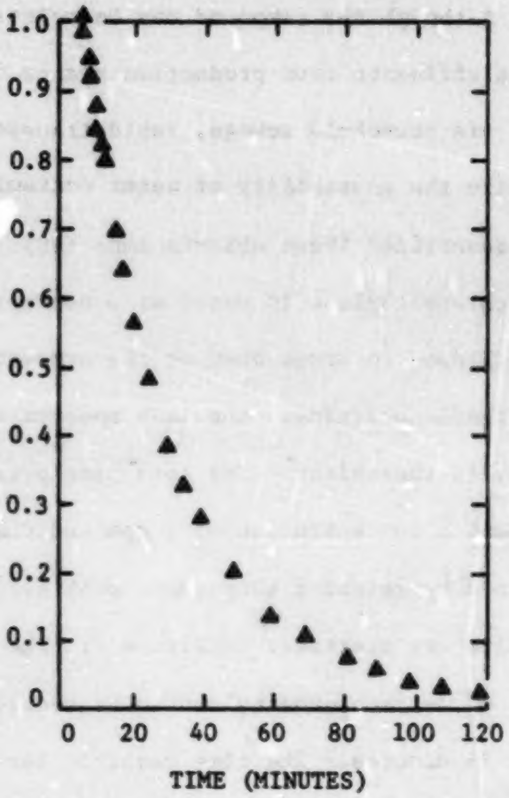
*Biological oxygen demand (five-day test): a measurement of oxygen uptake by microorganisms as a result of their action on a test chemical. These data are useful when estimating the lifetime of a molecule in aquatic or soil environments.

individually tested using a variety of parameters. These studies are being conducted by B. Gutnik and C. Xintaras at the U.S. Department of Health, Education and Welfare, Public Health Service Center for Disease Control, Cincinnati, Ohio.

C. AMBIENT CONCENTRATIONS, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY

Due to its high volatility and low solubility, tetrachloroethylene is expected to enter the environment primarily by evaporation to the atmosphere (59). Although the compound may be released to the hydrosphere via aqueous effluents from production plants or consumer industries and even via household sewage, rapid transport to the atmosphere will minimize the possibility of water contamination. A recent laboratory study quantified these observations (60). In the study, a solution of tetrachloroethylene in water at a concentration of 1 ppm was prepared and allowed to stand open to the atmosphere at approximately 25°C with gentle stirring. The mass spectra were scanned at zero time and at intervals thereafter. The zero time peak height was considered to represent a concentration of 1 ppm and changes in concentration were determined by relating subsequent peak heights to the zero time value. Results are presented in Figure 2. The time required for the concentration of tetrachloroethylene to be reduced by 50 percent was approximately 26 minutes. The time required for a 90 percent reduction was 83 minutes. With no stirring, the time required for 50 percent evaporation was 90 minutes. To obtain more ecologically significant data, the evaporation of tetrachloroethylene under conditions more nearly like those found in the environment was studied. The presence of 3 percent NaCl (as in sea water) caused about a 10 percent decrease in the evaporation rate after 40 percent had already evaporated. The

FIGURE II
EVAPORATION RATE OF $CC1_2 = CC1_2$ (▲) FROM WATER



Source: W. L. Dilling, N. B. Tefertiller and G. J. Kallos "Evaporation Rates and Reactivities of Methylene Chloride, Chloroform, 1,1,1-Trichloroethane, Trichero-ethylene, Tetrachloroethylene and other chlorinated compounds in dilute aqueous solutions." Environmental Science and Technology 9 833-837 (1975).

effect was, therefore, slight. The addition of 500 ppm of pre-soaked bentonite clay had no effect on the evaporation rate; addition of an equal amount of dry clay appeared to increase the rate of disappearance to 65 percent solute depletion at 20 minutes. A sealed system experiment in which solute loss could only occur by adsorption revealed that 305 ppm bentonite clay adsorbed 10 percent of the dissolved tetrachloroethylene in 10 minutes; 750 ppm adsorbed 22 percent after 30 minutes. Addition of 500 ppm dry dolomitic limestone caused a 50 percent depletion in 20 minutes and a 90 percent depletion in 70 minutes, indicating that tetrachloroethylene is also adsorbed slightly by limestone. Ottawa silica sand at 500 ppm had no effect on the disappearance rate. Peat moss (500 ppm) added to simulate a high aqueous organic content accelerated the disappearance initially, but appeared to slow it down at the later stages of the experiment (90 percent depletion in 120 ±15 minutes). In a sealed system, peat moss was found to remove 40 percent of the dissolved tetrachloroethylene in 10 minutes, accounting for the rapid initial disappearance; it is possible that a gradual release from the peat moss back to the solution occurred in the later stages of the study.

Propylene glycol (15 ppm) added to simulate a chemical plant waste effluent had no effect on the evaporation rate; however, addition of 1 ml of benzene to the surface of the water decreased the evaporation rate by 47 percent.

A 2.2 mph wind across the surface of the water increased the evaporation rate by 17 percent; reduction of the temperature to 1 to 2°C resulted in a 28 percent decrease in rate after 30 minutes, compared to that at 25°C.

Evaporation of tetrachloroethylene from the hydrosphere is, therefore, a rapid process, even in the presence of adsorbing contaminants. Tetrachloroethylene was recently identified as a component of the drinking water in New Orleans, Louisiana. Although concentrations were not specified, they were most likely lower than those used in the above study. It is difficult, on the basis of the data presented, to predict the rates of loss from water at much lower concentrations since the amount of oxidation and free air space above the water is not known.

Another parameter which must be considered when evaluating the potential persistence of a contaminant in a given compartment of the environment is the decomposition rate under environmental conditions. The rate of decomposition of tetrachloroethylene in aerated water both in the dark and in the presence of sunlight has been determined (60). The results are presented in Table VI. The rate constant for decomposition assuming a first order reaction was 0.079 months $^{-1}$, with a half-time of 8.8 months. Due to the high volatility of this compound and the large excess of oxygen in the air space of the sealed tube above the solution, it is possible that part of the reaction occurred in the vapor phase. Sunlight had a significant effect on the decomposition rate, indicating that disappearance was attributable largely to oxidation and was probably free-radical in character (60).

TABLE VI

DECOMPOSITION RATES OF TETRACHLOROETHYLENE IN THE DARK AND IN THE PRESENCE OF SUNLIGHT

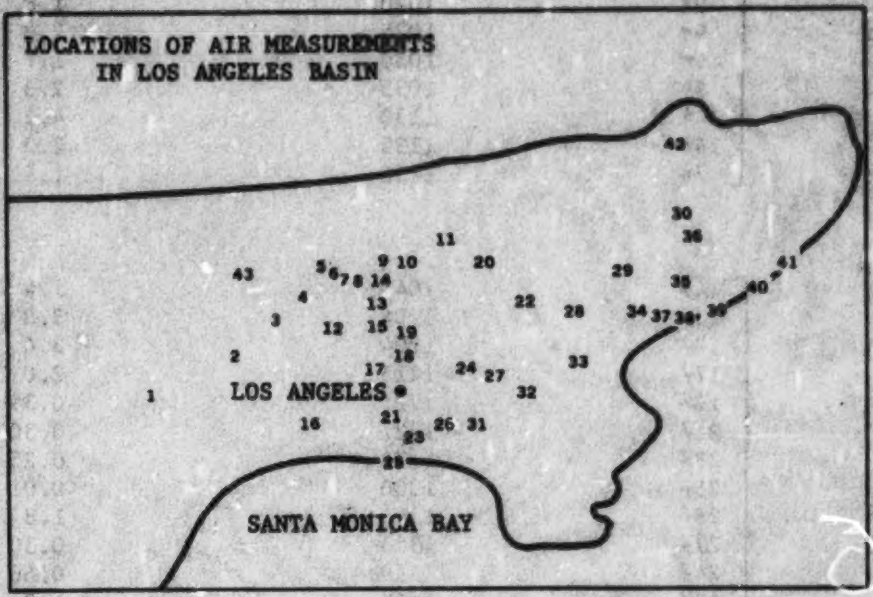
Time (months)	Concentration (ppm)	
	Dark	Light
0	1.00	1.00
6	0.63	0.52
12	0.38	0.25

Source: Dilling, W.L., N.B. Tefertiller, and G.J. Kallos. "Evaporation Rates and Reactivites of Methylene Chloride, Chloroform, 1, 1, 1 - Trichloroethane, Trichloroethylene, Tetrachloroethylene and Other Chlorinated Compounds in Dilute Aqueous Solutions." Environmental Science and Technology 1, 833-837 (1975).

Approximately 6.5 x 10^8 pounds (3.25 x 10^5 tons) of tetrachloroethylene were consumed in the United States in 1974 and it is estimated that 85 percent (2.76 x 10^5 tons) of this amount was released to the environment, almost entirely to the atmosphere (62). Aerial transport is also expected to provide the primary mode of dissemination throughout the biosphere. Since the vapor density of tetrachloroethylene is 5.5 times greater than that of air, under stagnant conditions the compound would tend to spread laterally as opposed to upward, resulting in distribution to areas far removed from the sources of contamination (2).

A recent study of the distribution of halocarbons in the air over the Los Angeles Basin provides data on the mechanism and extent of air transport of tetrachloroethylene (63). Ambient measurements of four halogenated hydrocarbons (including tetrachloroethylene) that are commonly used as solvents were made at various locations throughout Los Angeles County. Locations are presented in Figure III. Tetrachloroethylene was observed at all sites sampled. Concentrations at each location are presented in Table VII. The first set of measurements (Group "a" on Table VII) was taken in the Pasadena area on a day when visible smog and generally stable inversion conditions existed. Of the four halocarbons observed, tetrachloroethylene was present overall in the highest concentrations (1.3 ppb to 4.2 ppb). The second group of measurements (Group "b" on Table VII) was taken along a line from the West San Gabriel Valley to Manhattan Beach. There was

FIGURE III
MAP OF THE LOS ANGELES BASIN
(Numbers correspond to locations referenced in Table VII)


LOCATIONS OF AIR MEASUREMENTS
IN LOS ANGELES BASIN
42
30
36
11
9
10
20
29
41
43
5
6
7
8
14
35
40
4
13
22
28
34
37
38
39
3
12
15
19
2
18
33
17
24
27
32
1
LOS ANGELES
16
21
26
31
23
25
SANTA MONICA BAY

Source: P. G. Simmons, S. L. Kerrin, J. E. Lovelock and F. H. Shair "Distribution of Atmospheric Halocarbons in the Air Over the Los Angeles Basin" Atmospheric Environment 8 209-216 (1974)

TABLE VII

CONCENTRATION OF TETRACHLOROETHYLENE AT VARIOUS LOCATIONS IN LOS ANGELES COUNTY

	Location of Measurement	Time	C_2Cl_4 (ppb)
Group "a"	7*	0950	1.3
	14*	1005	2.5
	14*	1015	2.1
	9*	1020	1.8
	9*	1025	2.2
	9*	1030	1.7
	9*	1035	2.3
	14*	1130	4.2
	14*	1135	2.3
	7*	1150	1.9
Group "b"	4†	1040	1.4
	12†	1104	3.8
	19†	1204	3.6
	17†	1215	2.0
	16†	1221	0.39
	21†	1227	0.30
	23†	1235	0.25
	25†	1300	<0.01
	26†	1345	1.8
	27†	1353	0.30
	24†	1359	0.66
	18†	1407	1.8
	19†	1410	1.4
	15†	1420	0.78
	13†	1426	0.15
	8†	1433	1.2
	7†	1440	1.1
	5†	1445	1.9

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* Date: 22 September 1972: Wind heading 210-250° 5-8 knots.

† Date: 28 September 1972: Wind heading 220-250° 8-10 knots.

TABLE VII (Continued)

	Location of Measurement	Time	C_2Cl_4 (ppb)
Group "c"	6+	0900	0.70
	10+	0915	1.13
	11+	0925	1.38
	20+	0935	2.77
	22+	0940	2.32
	29+	0950	0.60
	30+	1000	0.93
	41+	1040	0.06
	40+	1050	3.84
	39+	1100	1.72
	38+	1105	1.98
	37+	1110	2.03
	34+	1115	0.75
	28+	1120	2.03
	29+	1125	2.71
	36+	1130	2.57
	30+	1145	1.15
	30+	1345	0.67
	35+	1400	0.97
	34+	1405	0.92
	33+	1415	0.37
	32+	1420	1.04
	31+	1430	0.96
	21+	1435	0.87
	16+	1440	0.44
	1+	1450	0.88
	2+	1458	0.48
	3+	1504	1.47
	7+	1512	1.38
	7+	1515	1.16
	42≠	24-hr. aver.	0.09

+ Date: 4 October 1972: Wind heading Var. 305-45° (0800). Calm (1000). 250° at 8 knots (1230).

≠ Date: 6 October 1972: San Bernardino Mountains - Average values for 24-hour sampling time.

Source: P.G. Simmons, S.L. Kerrin, J.E. Lovelock and F.H. Shair. "Distribution of Atmospheric Halocarbons in the Air Over the Los Angeles Basin." Atmospheric Environment 8,209-216 (1974).

a progressive decrease in the concentration upon moving from the inland valley to the coast, interrupted only by high concentrations of tetrachloroethylene in the Los Angeles business district, presumably caused by local emissions. Inland valley concentrations ranged from 0.78 to 1.2 ppb (average of 1.11 ppb); concentrations in and around Los Angeles ranged from 1.4 to 3.8 ppb (average of 2.52 ppb), and concentrations along the coast ranged from 0.25 to 1.8 ppb (average of 0.61 ppb). The lowest concentration of tetrachloroethylene (<0.01 ppb) was measured in the maritime air flowing directly off the ocean with a wind speed of 10 knots. Tetrachloroethylene in the air near or at the cost was most likely of airborne origin.

The third set of measurements (Group "c" on Table VII) covered a wider geographical area and was obtained during variable meterological conditions. These conditions were marked by a midday reversal in the wind direction. Unusually high concentrations were observed at this time at several locations (sites 38-41) along the coast in Orange County. Concentrations ranged from 0.96 to 3.89 ppb (average of 2.13 ppb). At the time of measurement, conditions were calm with no offshore wind. But prior to these measurements, the prevailing wind was reported from the NW-NE. It seems reasonable to assume that a polluted air mass may have accumulated over Orange County during the early morning and had not dissipated at the time of measurement. When the wind direction changed to SSW at 10 knots, a general reduction in concentration at all sites in the area was noted. The average concentration of tetrachloroethylene throughout the entire Los

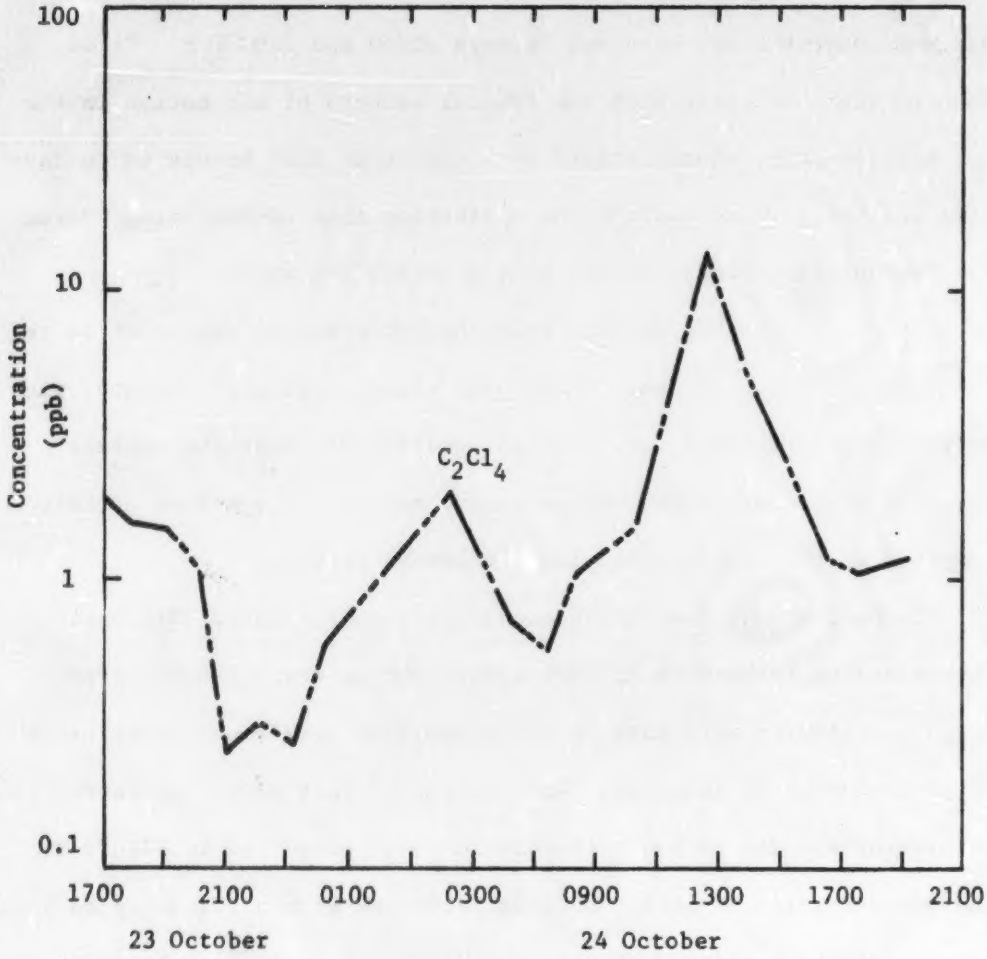
Angeles Basin was 1.25 ppb.

A series of measurements was also obtained for a 24-hour period in the West San Gabriel Valley (Location 43, Figure III). Results of this diurnal study are presented in Figure IV. It can be seen that the maximum concentration occurred between 12:00 and 1:00 P.M. These results are consistent with the typical pattern of air motion in the Los Angeles area, characterized by a nighttime land breeze and a daytime sea breeze, and confirm the contention that tetrachloroethylene is transported through the air by the prevailing winds.

It is interesting to note that the direction of transport in the evening is out toward sea. Therefore, slow-reacting pollutants, such as tetrachloroethylene, are free to equilibriate with the surface layer of the ocean which, consequently, may act as a source of this compound in the morning when the sea breeze prevails.

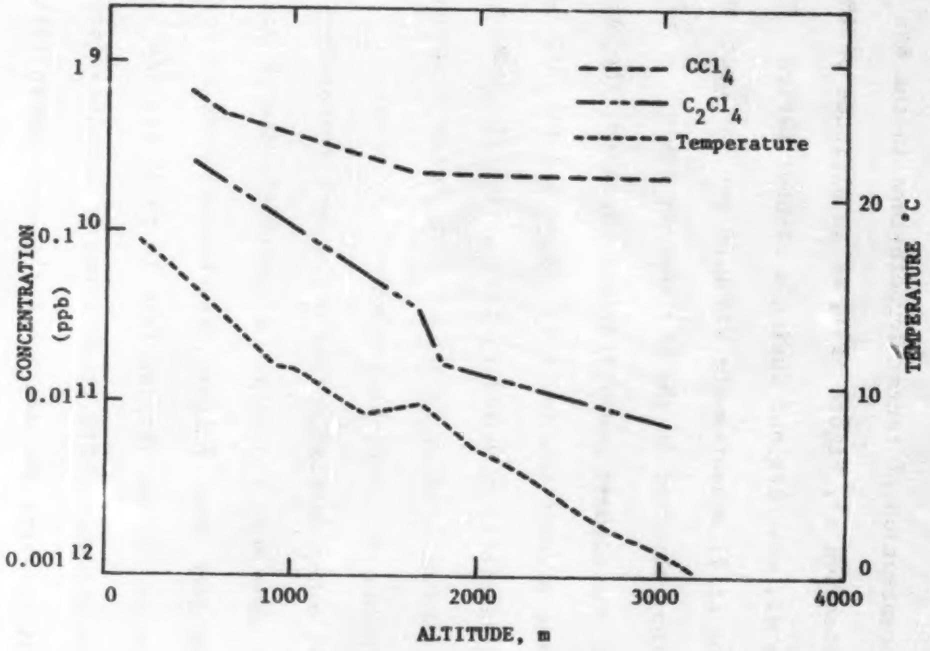
Concentrations over Inglewood (Location 21, Figure III) were determined up to heights of 3200 meters during several helicopter flights. Efforts were made to avoid sampling near known major users of the solvents of interest. The results of this study for tetrachloroethylene and carbon tetrachloride are presented in Figure V. The concentration of both compounds decreased with altitude up to 1900 meters, at which point a significant inversion layer was observed. Above this inversion level, the concentration of carbon tetrachloride remained constant (at 0.23 ppb). Tetrachloroethylene concentration, however, continued to decrease with altitude. The authors feel that

FIGURE IV
CONCENTRATION VS. TIME DATA FOR C_2C1_4 AT GROUND LEVEL AT LOCATION 43 IN THE WEST SAN GABRIEL VALLEY



Source: P. G. Simmons, S. L. Kerrin, J. E. Lovelock and F. H. Shoir. "Distribution of Atmospheric Halocarbons in the Air Over the Los Angeles Basin." Atmospheric Environment 8 209-216 (1974)

FIGURE V
TYPICAL CONCENTRATIONS OF HALOCARBONS AND AMBIENT TEMPERATURE VS. ALTITUDE OVER INGLEWOOD
(location 21)



Source: P. G. Simmons, S. L. Kervin, J. E. Lovelock and F. H. Shoir. "Distribution of Atmospheric Halocarbons in The Air Over the Los Angeles Basin." Atmospheric Environment 8, 209-216 (1974).

these results imply that the existing concentration of tetrachloroethylene represents emissions from stationary sources in the Los Angeles Basin, whereas an atmospheric background level of carbon tetrachloride must be present.

The concentration of tetrachloroethylene in the San Bernadino Mountains (Location 42, Figure III), at an altitude of 1800 meters above sea level, was obtained during a 24-hour period. The average concentration of 21 measurements was 0.09 ppb, an order of magnitude lower than that observed in the Los Angeles Basin.

Even at the highest concentrations observed, the amount of tetrachloroethylene present was too small to constitute any health hazard. There was a possibility, however, that during the summer months when stagnant inversion conditions are present, often on consecutive days, a greater buildup of tetrachloroethylene may occur.

Several other determinations of ambient tetrachloroethylene concentrations have been reported. A systematic search for simple halocarbons made over rural Pullman, Washington revealed tetrachloroethylene concentrations varying from 10 to 30 ppt (64). These values were in good agreement with those found by Lovelock (65) in 1973 in the North Atlantic, where the average background concentration was 21 ppt. Concentrations measured in the air over western Ireland (27.6 ppt) by Lovelock were also the same order of magnitude (65).

The presence of tetrachloroethylene in virtually all compartments of the biosphere testifies to its widespread dissemination at generally low concentrations (1 part in 10^9). Typical values from various sources are presented in Table VIII.

Since the compound is fairly rapidly degraded in the environment and no evidence of significant bioaccumulation exists, tetrachloroethylene does not represent a significant pollution threat to the general population. The highest concentrations to which an average person might be exposed would be expected to occur in the vicinity of production plants or dry cleaning operations. Although ambient concentrations of tetrachloroethylene near production facilities have not been reported, it is possible to estimate such concentrations using production figures along with certain basic assumptions.

Concentrations of tetrachloroethylene outside the plant can occur through the ventilation and exhaust of internal air. Atmospheric dispersion would distribute the gaseous emissions downwind as affected by the turbulent diffusion of the air. Hypothetical plant conditions could be used as inputs to a Gaussian plume equation* from Turner's

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*Ground-level downwind concentrations resulting from a point source are predicted by the following equation:

$$\chi(x, 0, 0; H) = \frac{Q}{\pi\sigma_y \sigma_z u} \exp\left[-1/2 \left(\frac{H}{\sigma_z}\right)^2\right]$$

where: Q = uniform emission rate (grams/sec)
u = mean wind speed affecting the plume (meters/sec)
H = effective stack height; that is, sum of stack height plus plume rise (meters)
σ_y = horizontal dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters)
σ_z = vertical dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters)
χ = ground-level concentration on plume axis (grams/meter^3)

TABLE VIII

OCCURRENCE OF TETRACHLOROETHYLENE IN THE ENVIRONMENT
(Typical concentrations (w/w))

	MINIMUM	MAXIMUM
Air	10^{-9}	10^{-8}
Rainwater	10^{-11}	10^{-9}
Surface water	10^{-11}	10^{-9}
Potable water	10^{-11}	10^{-9}
Sea water	10^{-10}	10^{-9}
Marine sediments	10^{-10}	10^{-9}
Marine invertebrates	10^{-9}	10^{-8}
Fish	10^{-9}	10^{-8}
Waterbirds	10^{-9}	10^{-7}
Marine mammals	10^{-9}	10^{-8}
Fatty foods	10^{-9}	10^{-8}
Non-fatty foods	10^{-9}	10^{-9}
Human organs	10^{-9}	10^{-9}
Human body fat	10^{-9}	10^{-8}

Source: McConnell, G., D. M. Ferguson, and C. R. Pearson, "Chlorinated hydrocarbons and the environment," Endeavor 34, 13-18, 1975.

Workbook of Atmospheric Dispersion Estimates (66). The basic diffusion equation should be modified, however, due to the effect of the plant itself on the flow of air. Mechanical turbulence in the wake of a building tends to produce aerodynamic downwash, resulting in the fairly rapid diffusion of a gaseous emission down to the ground. This region of disturbed flow extends downwind, a distance equal to several times the height of the building. While this situation does not result in a Gaussian-distributed plume, it is proposed that a modification of the usual formula still can be employed. This is because the turbulent mixing in the wake of the building is assumed to be distributed uniformly in the vertical direction, analogous to the situation where a plume is trapped below an inversion layer. A limited vertical mixing height can be modeled using the equation:

$$\chi(x,\ 0,\ z;\ H) = \frac{Q}{\pi\sigma_y u\ (.8L)}$$

where L is the limit of the mixing depth. In the case of an isolated rectangular building, it is assumed that L equals 1.5 times the building height.

In addition, there is a horizontal wind turbulence which is assumed to result in an initial horizontal plume spread equal to the width of the building normal to the wind direction. This is analogous to an area source emission where the area source is the building top. This can be modeled using a further modification of the basic Gaussian diffusion equation. A virtual point source is assumed upwind from the building at a distance where the plume spread (for the given stability conditions) would equal the crosswind of the building. The modified diffusion equation is therefore:

$$(x,0,z;\ H) = \frac{Q}{\pi \sigma_y' u\ (1.2h)}$$

where H is the building height and σ_y' is based on the downwind distance to the receptor point, plus the upwind distance to the virtual point source.

If it can be assumed that a plant produces the maximum expected output of 200 million pounds (10^5 tons) of tetrachloroethylene annually, with a production loss of 10 percent and a treatment efficiency of 98 percent obtained through the use of some emission control method, then 21.4 grams per second would be emitted to the atmosphere, where the plant operates 360 days a year, 24 hours a day. At this emission rate, the ambient concentration 500 meters downwind from the plant would be 406 $\mu g/m^3$, or 0.06 ppm, as estimated in the modified diffusion equation.* The downwind distance of 500 meters from the source was chosen to represent a point within the perimeter of a plant, and ambient concentrations in surrounding neighborhoods could be expected to be significantly lower.

An individual standing 500 meters downwind from a production facility would have to remain there for 380 years in order to inhale an amount equivalent to that inhaled by an individual worker occupationally exposed to 100 ppm over an 8-hour workday.

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*Additional assumptions: (1) tetrachloroethylene is nonreactive; (2) building height is 50 feet (15.24 meters), is 100 feet (30.48 meters); (3) the atmospheric stability class is neutral; (4) wind speed is 6 meters/sec.

The population incurring the highest risk of exposure to unhealthy levels of tetrachloroethylene are the industrial workers in daily contact with this solvent. The present acceptable levels are 300 ppm for a "peak" of not more than 5 minutes duration, 200 ppm for a "ceiling," and 100 ppm for a time-weighted average over an 8-hour workday (19). While adverse symptoms definitely occur at 200 to 300 ppm, evidence exists that exposure to 100 ppm is not entirely without effect (20). In 1974, as a result of complaints from workers, OSHA's main analytical laboratory in Salt Lake City, Utah, analyzed 51 industrial atmospheric samples for tetrachloroethylene (67). The results of these analyses were sent to the appropriate District Office, but a national compilation of the results has not been published.

Techniques for the isolation, identification, and measurement of halogenated hydrocarbons have been extensively studied. Several methods of collection are used (19). Air samples may be collected in plastic bags (Saron or Mylar are acceptable) and analyzed by any appropriate method, such as infrared spectroscopy, gas chromatography, or mass spectrometry. Samples absorbed into a solvent can be determined by the Fujiwara reaction (37): tetrachloroethylene in a solution is decomposed chemically, or it may be decomposed by combustion, and subsequently absorbed and measured as chloride ion. Samples absorbed from an airstream onto a solid, such as silica gel or activated carbon, must be removed either by a solvent (such as carbon disulfide) or by an air or nitrogen stream passed over the heated solid.

Gas chromatography using an election capture detector or flare ionization detector has been successfully employed for the analysis of tetrachloroethylene (63,68). However, highest sensitivity (5 ppt) and precision (±5 percent) are afforded by using a gas chromatograph in conjunction with a mass spectrometer (64).

An air sampling device particularly suited for periodic monitoring of work areas employs a glass tube packed with activated charcoal and eliminates the need for bulky equipment, such as the Davis Halide Meter (68). Analysis of the exhaled air of workers is a useful measure of past exposure levels. Infrared spectroscopy may be used for qualitative determination when an unknown mixture of vapor is involved, whereas quantitative results may be obtained with gas-liquid chromatography (19).

D. SOURCES

Faraday first prepared tetrachloroethylene in 1821 by thermal decomposition of hexachloroethane. Industrial production began in the United Kingdom and in Germany shortly before World War I, but did not begin in the United States until 1925. Subsequently, demand for the solvent increased domestically with the introduction of the small dry cleaning unit. By the end of World War II, tetrachloroethylene had displaced carbon tetrachloride from its dominance in the domestic dry cleaning industry. In addition, it was successfully competing with trichloroethylene in the United Kingdom. Tetrachloroethylene now ranks second in production (to trichloroethylene) among the chlorinated solvents (1).

Prior to 1970, tetrachloroethylene was produced by one of several methods. With acetylene as a starting material, pentachloroethane was synthesized and dehydrohalogenated to the desired product. Direct chlorination of acetylene to tetrachloroethylene was also employed. Using tetrachloroethane as a starting material, tetrachloroethylene was produced either by chlorination (with HCl as a side product) or by oxidation (with water as a side product).* Other methods include direct chlorination of ethane or ethylene (1). By 1970, 80 percent of the tetrachloroethylene production proceeded via the thermal chlorination of propane:

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*$CHCl_2 - CHCl_2 + Cl_2 \longrightarrow CCl_2 = CCl_2 + 2HCl$

$2CHCl_2 - CHCl_2 + O_2 \longrightarrow 2CCl_2 = CCl_2 + 2H_2O$

$$C_3H_8 + 8\ Cl_2 \xrightarrow{\hspace{4em}} CCl_4 + C_2Cl_4 + 8\ HCl$$

The remaining 20 percent was produced from acetylene as described above (62, 70). In 1972, the two major acetylene-based plants in the United States were closed and it is expected that in the future all tetrachloroethylene will be synthesized from propane.

There are eight producers of tetrachloroethylene in the United States; the location of their plants and their plant production capacities as of January 1975 are presented in Figure VI and Table IX. Production and sales figures from 1940 to 1972 are presented graphically in Figure VII. In the period from 1960 to 1970, annual production increases averaged 12 percent. For the current decade, an annual production growth rate of about 7 percent is expected (3.67×10^5 tons) (3). The world production capacity as of 1972 was estimated to be 1.9 billion pounds per year (9.5×10^5 tons/yr) and is estimated at present to be about 2 billion pounds per year (1×10^6 tons/yr). By 1980, worldwide demand for tetrachloroethylene is expected to reach 2.4 billion pounds per year (1.2×10^6 tons/yr) (70).

Imports into the United States increased from 22.1 million pounds (1.11×10^4 tons) in 1960 to a high of 70 million pounds (3.5×10^4 tons) in 1964 (70). They have fluctuated considerably since that time and, as of 1973, amounted to 26.7 million pounds (1.34×10^4 tons) (3). Exports during 1973 amounted to 112.6 million pounds (5.63×10^4 tons) (3), but are expected to decline due to the startup of Dow's plant at Stade, West Germany (70).

FIGURE VI
LOCATIONS OF UNITED STATES TETRACHLOROETHYLENE PRODUCTION FACILITIES

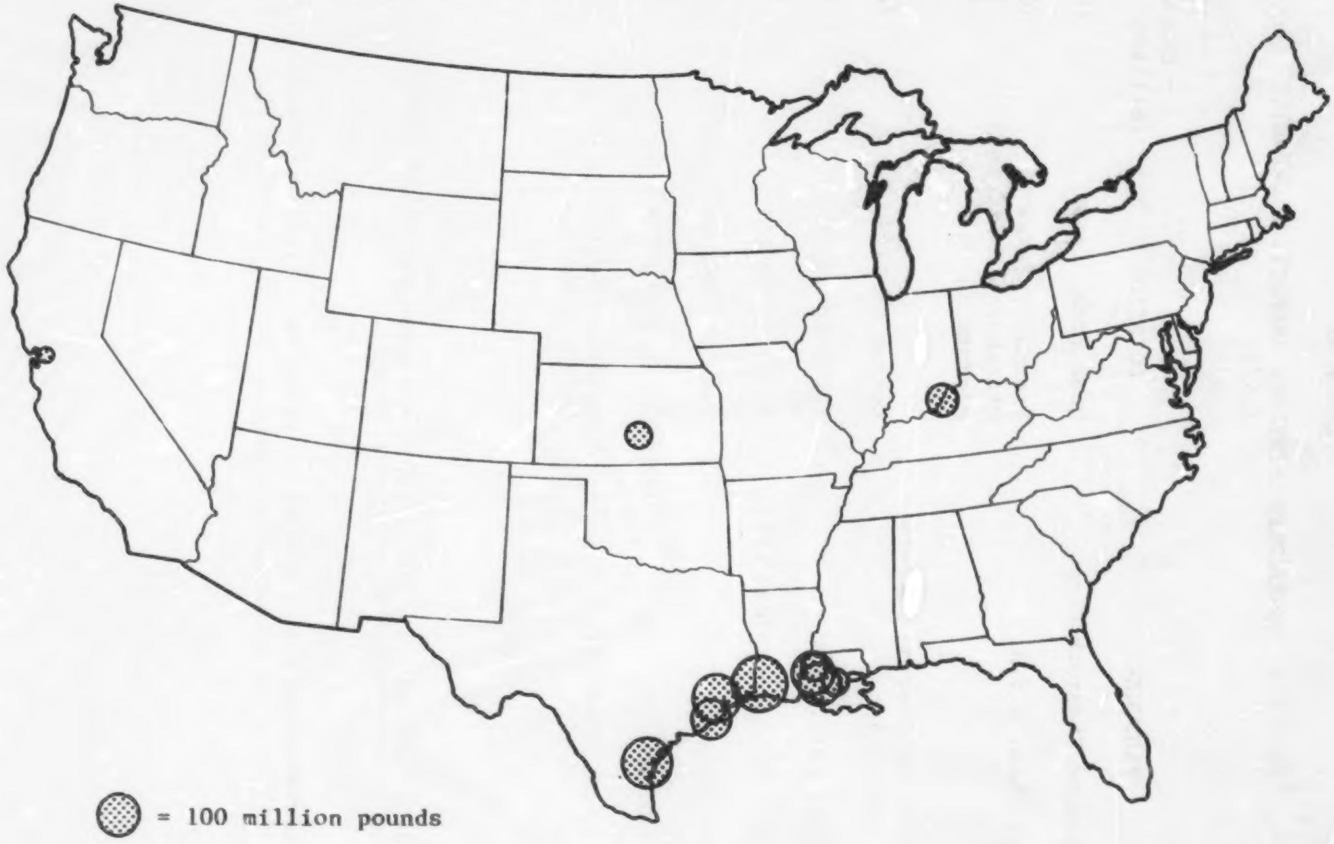
= 100 million pounds

TABLE IX

U.S. TETRACHLOROETHYLENE PRODUCTION CAPACITY[1] (1975)

PRODUCER	LOCATION	CAPACITY[1] (millions of pounds)
Diamond Shamrock Corp.	Deer Park, Texas	160
Dow Chem. U.S.A.	Freeport, Texas	120
	Pittsburg, California	20
	Plaquemine, La.	150
E. I. du Pont de Nemours & Co., Inc.	Corpus Christi, Texas	160
Ethyl Corp.	Baton Rouge, La.	50
Occidental Petroleum Corp.	Taft, Louisiana	50
PPG Indust., Inc.	Lake Charles, Louisiana	200
Stauffer Chem. Co.	Louisville, Kentucky	70
Vulcan Materials Co.	Geismar, Louisiana	150
	Wichita, Kansas	50
TOTAL		1180

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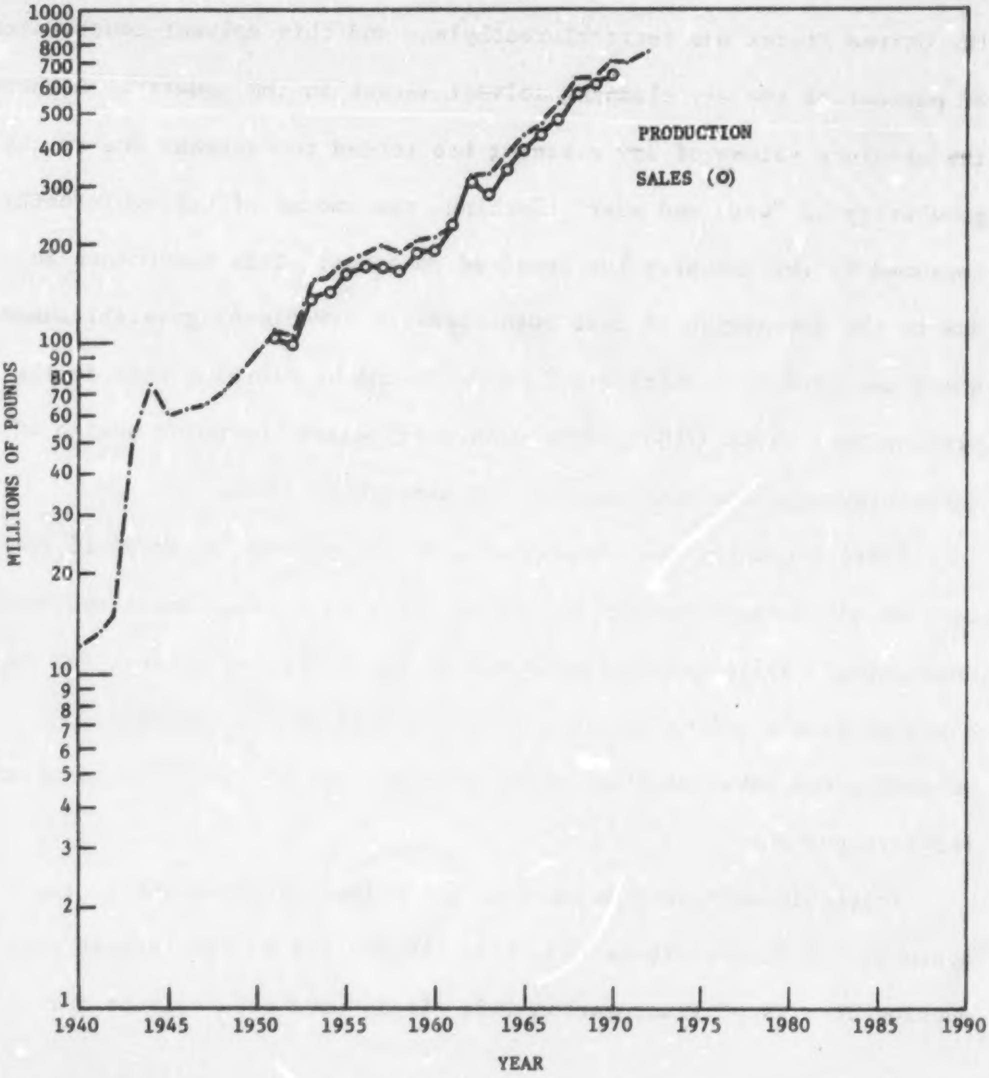
[1] As of January 1, 1975. Capacities are extremely flexible, depending on product balance.

Source: Chemical Information Services, Stanford Research Institute. 1975 Directory of Chemical Producers - USA Page 725.

FIGURE VII
PRODUCTION AND SALES OF TETRACHLOROETHYLENE
1940-1972



Source: Blackford, J. L., "Perchloroethylene," Chemical Economics Handbook, Stanford Research Institute, Menlo Park, California, 685. 5081A-685. 5032X.

About 60 percent of domestic consumption of tetrachloroethylene is by the textile industry, primarily for dry cleaning but also for processing and refinishing. About 55 percent of the dry cleaners in the United States use tetrachloroethylene and this solvent constitutes 80 percent of the dry cleaning solvent market in the country. Although the absolute volume of dry cleaning has tended to decrease due to the popularity of "wash and wear" clothing, the amount of tetrachloroethylene consumed by the industry has remained constant. This phenomenon is due to the appearance of more coin-operated dry cleaning establishments, which use more tetrachloroethylene per pound of clothing than do the professional shops (70). These plants represent the major source of tetrachloroethylene emissions to the atmosphere (71).

Tetrachloroethylene is also used as the solvent in about 15 percent of all metal-cleaning operations for both cold cleaning and vapor degreasing. While trichloroethylene is the preferred solvent for vapor degreasing, air pollution restrictions placed on this solvent in several areas have resulted in an increased use of tetrachloroethylene for this purpose.

Tetrachloroethylene is used as a chemical intermediate in the synthesis of Fluorocarbons 113, 114, 115 and 116.* The largest production item is of Fluorocarbon 113. It is used as a solvent for

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*Fluorocarbon 113 - trichlorotrifluoroethane
Fluorocarbon 114 - dichlorotetrafluoroethane
Fluorocarbon 115 - chloropentafluoroethane
Fluorocarbon 116 - hexafluoroethane

photographic films and magnetic tapes, for cleaning plastic materials, electrical and electronic equipment, as a dry cleaning agent, as a refrigerant, and as an intermediate in the production of chlorotrifluoroethylene monomer. Fluorocarbon 114 is used as a refrigerant, solvent, and aerosol, while Fluorocarbon 115 is used as a food propellant. The production of Fluorocarbon 116 is very small and it is used as a dielectric gas (70).

Miscellaneous uses account for only a small percentage of the total tetrachloroethylene production. These include solvent for silicones, aerosol specialty products for laundry treatment (70), and pharmaceutical products (anthelmintic medicine) for killing or ejecting intestinal worms (5,6,35,36). Table X shows the percent distribution of tetrachloroethylene by use. A flow chart summarizing production, consumption, import/export, and loss figures is presented in Figure VIII.

TABLE X

CONSUMPTION OF TETRACHLOROETHYLENE*

USE	PERCENT
Dry Cleaning, Processing and Finishing of Textiles	58
Exports	17
Solvent for Metal Cleaning	15
Manufacture of Other Chemicals	9
Miscellaneous	1

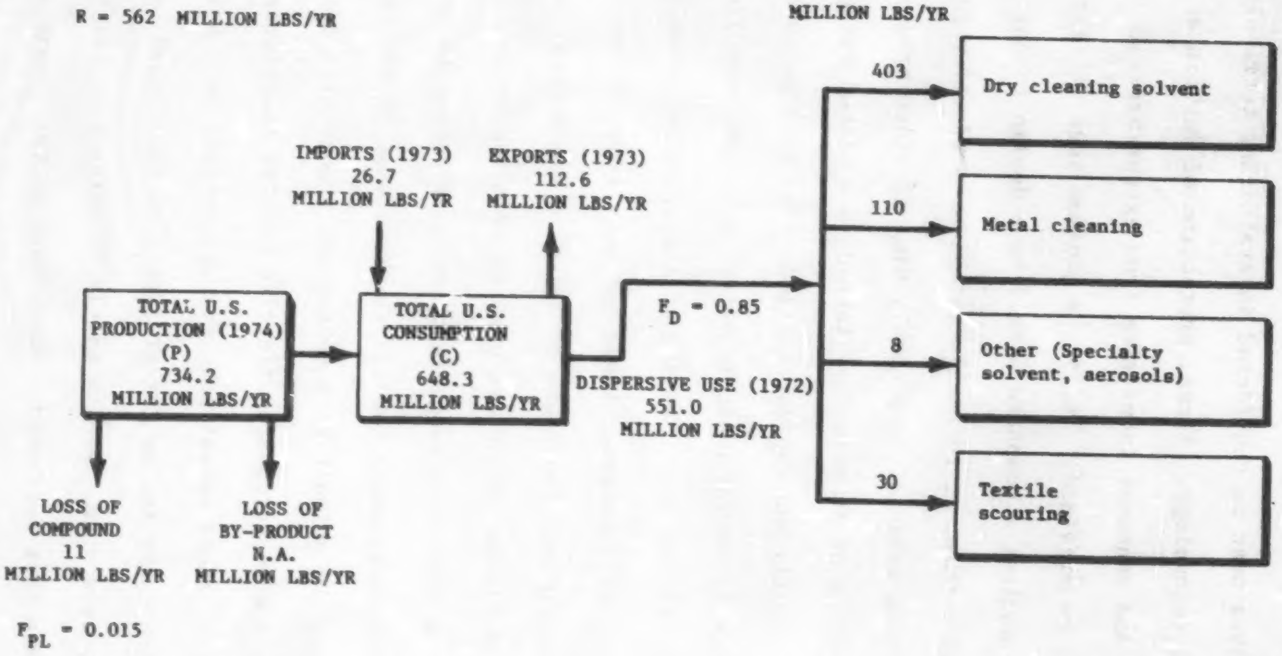
* 1971 estimate.

Source: Blackford, J.L., "Perchloroethylene," Chemical Economics Handbook, Stanford Research Institute, Menlo Park, California, 685.5081A-685.5032X.

FIGURE VIII
SUMMARY OF PRODUCT RELEASE INFORMATION

Tetrachloroethylene

R = 562 MILLION LBS/YR


MILLION LBS/YR
TOTAL U.S. PRODUCTION (1974) (P) 734.2 MILLION LBS/YR
IMPORTS (1973) 26.7 MILLION LBS/YR
EXPORTS (1973) 112.6 MILLION LBS/YR
TOTAL U.S. CONSUMPTION (C) 648.3 MILLION LBS/YR
F_D = 0.85
DISPERSIVE USE (1972) 551.0 MILLION LBS/YR
403
Dry cleaning solvent
110
Metal cleaning
8
Other (Specialty solvent, aerosols)
30
Textile scouring
LOSS OF COMPOUND 11 MILLION LBS/YR
LOSS OF BY-PRODUCT N.A. MILLION LBS/YR
F_{PL} = 0.015

Source: Brown, S. L., F. Y. Chan, J. L. Jones, D. H. Liv, K. E. McCaleb, T. Mill, K. N. Sapios, and D. E. Schendel, "Research Program on Hazard Priority Ranking of Manufactured Chemicals," SRI Project ECU-3386. Stanford Research Institute, Menlo Park, California, 1975.

R = rate of loss (production and dispersive use)
F_{PL} = fraction of production loss
F_D = dispersion loss.

E. CONTROL

Two objectives must be considered in evaluating tetrachloroethylene control technology. First, protection of workers at production facilities and consumer industries from excess exposure to hazardous levels is to be provided. Second, a minimum loss of tetrachloroethylene to the ambient atmosphere should be a design factor.

Exposure of workers at tetrachloroethylene production plants using propane as starting material is probably minimal since production by thermal chlorination of propane is effected in a closed system, with all nonproduct liquids and vapors recycled. The only pollution source is a purge to remove inert gases prior to the recycling of remaining chlorine to the feed. Drying of the chlorine takes place after raw product tetrachloroethylene has been removed from the system (69). Assuming that there are no leaks in the system, negligible amounts of tetrachloroethylene are emitted from the production process.

The major source of tetrachloroethylene emissions in dry cleaning is the tumbler through which hot air is circulated to dry the clothes. Drying vaporizes the solvent with consequent atmospheric emissions. Condensation and adsorption are effective control techniques used together to control these emissions. Primary control is a water-cooled condenser, which comprises an integral part of the closed cycle in a tumbler or drying system. Approximately 95 percent of the vapors from the clothing are thus recovered. About half of the remaining solvent is then captured with an activated-carbon adsorber. Overall control efficiency is 97 to 98 percent. However, only 25 percent of dry cleaning plants using tetrachloroethylene employ this emission control.

During the process of "cold cleaning" of metals near ambient temperatures, several precautions may be taken to minimize worker exposure (72). Loss to the atmosphere may be reduced by using lids or plunger containers, and by closing lids on tanks or containers when the tank is not in use or when parts are soaking. Lids should be opened slowly, since fast opening tends to withdraw vapors. Tanks should be placed away from doors, paint spray booths, overhead heaters, or ventilators. Baffles should be placed on the windward side of a tank when an ideal location is not available. An external cooling jacket should be placed on tanks. In summertime, cooling coils immersed in the tank should be used to avoid excessive solvent vaporation.

For vapor degreasing, alternate solvents to tetrachloroethylene exist and substitution might be considered if operationally and economically acceptable. However, careful evaluation of the health hazards which may result from use of the substitute material must be provided. One possibility is an inhibited grade of 1,1,1-trichloroethane, which has been demonstrated to be the most easily controlled vapor-degreasing solvent under OSHA regulations (72).

If tetrachloroethylene must be used, cooling coils or other vapor reclaiming devices should be added to the unit. Heat should be adjusted so that the vapor level idles at the midpoint of the cooling coils and cooling water temperature should be adjusted to 100 to 110°F as it exits from the unit. Heat input must balance condensing capacity and radiation losses. Air currents should be regulated so as to prevent drafts across the open area of the unit; disturbance of the vapor may be minimized by avoiding the opening of windows, turning on of fans, etc., near the degreasing operation. Local exhaust ventilation should be installed as close as possible to the point where the vapor escapes or to the source of contamination, and should be designed to exhaust vapors from the workers' breathing zone (72). Excessive water in the solvent lowers the vapor density, which results in excessive solvent loss. Water separators of efficient design should be installed which will continuously eliminate any water that gets into the system. Care must be taken not to overload the unit and cause an excessive drop of the vapor level. Air displacement and replacement occurs as a result of overloading. A mixture of air and solvent vapor is expelled as the vapor level returns to the correct operating height. Finally, workloads should be left in the degreaser until they reach vapor temperatures. Parts will then be dry when removed and eliminate the necessity of secondary ventilation to control vapor emissions from the work.

In summary, for any process using tetrachloroethylene, proper equipment design, adequate general ventilation, and prompt cleanup of leaks, spills, and waste recepticals contribute effectively to maintaining safe vapor levels in the workshop.

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